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ABSTRACT

At each meeting of the Pan American Health Organization Advisory Committee on Medical Research, a special 1-day session is held on a topic chosen by the committee as being of particular interest. At the 7th meeting, which convened in June of 1968 in Washington, D.C., the session surveyed the origin, present distribution, and principal biological and medical issues of the American Indian--including the problems of newly contacted Indian groups and those of groups well along in transition. Recorded in this volume are the following papers and ensuing discussions: (1) The Origin and Dispersion of American Indians in North America; (2) Theses for Meditation on the Origin and Dispersion of Man in South America; (3) Biological Subdivisions of the Indian on the Basis of Physical Anthropology; (4) Biological Subdivisions of the Indian on the Basis of Genetic Traits; (5) The American Indian in the International Biological Program; (6) Survey of the Unacculturated Indians of Central and South America; (7) Medical Problems of Newly Contacted Indian Groups; (8) The Problem of Gallbladder Disease among Pima Indians; (9) Hyperglycemia in Pima Indians; (10) Malaria in the American Indian; (11) Food and Nutrition of the Maya Before the Conquest and at the Present Time; (12) Iodine Deficiency Without Goiter in Isolated Yanomama Indians; and (13) Study of Endemic Goiter in the American Indian. (Author/LS)

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BIOMEDICAL CHALLENGES PRESENTED BY THE AMERICAN INDIAN

**Proceedings of the Special Session
held during the Seventh Meeting of the
PAHO Advisory Committee
on Medical Research
25 June 1968**

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NOTE

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BIOMEDICAL CHALLENGES PRESENTED BY THE AMERICAN INDIAN

Moderator: Dr. James V. Neel

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OPENING STATEMENT

James V. Neel, Moderator

The American Indian continues today, as he has ever since the discovery of the Americas, to present a wide variety of questions and challenges ranging from the purely scientific to the intensely humanitarian. In today's Special Session, we shall be able to consider only a few of these many challenges.

Specifically, we shall deal first with some of the unusual opportunities the Indian still offers for the investigation of basic problems in genetics and anthropology. These opportunities include studies of the nature and rate of human dispersion, of the emergence of human diversity, and of the biology of primitive man, with all its ramifications.

In the latter half of the program, we shall turn our attention to some of the more medical and humanitarian problems presented by the Indian. In many respects, these problems are similar to those of the *campesino* or *caboclo* throughout South America, to whose gene pool the Indian has made a significant contribution.

However, the Indian also presents some special problems. One of these is the transition, in the relatively unacculturated Indian, from "small band" epidemiology to "large herd" epidemiology. As we are all aware, this transition has repeatedly had a fatal impact on Indian groups.

The medical challenge to blunt this impact is clear and persistent. In addition, by taking it up we preserve at least temporarily one of the last great resources for the study of primitive man, one of the last opportunities to attempt to fathom the nature of the forces to which man was responding during the course of human evolution. Further, it begins to appear as if once that transition is accomplished the Indian may present some rather special disease problems, whose satisfactory solution will challenge all our medical ingenuity.

There is one important biomedical problem of the Indian that this session will leave completely untouched—the problem of high-altitude populations. This subject was covered both at the Special Session of 1966[1] and at a WHO/PAHO/IBP conference held here in November 1967. Under these circumstances, a further brief treatment at this time was deemed redundant.

The number of participants in the Session is somewhat greater than usual, thanks to the generous financial assistance of both the U.S. National Committee for the International Biological Program and the Human Adaptability Section of the Committee for the International Biological Program of the International Union of Biological Sciences.

Today's formal presentations will be followed tomorrow by an informal session at which problems of common interest to the participants here today will be discussed, with particular reference to the International Biological Program.

It is customary at such a session to designate two rapporteurs. This year Dr. Napoleon Chagnon and Dr. Edmund Covarrubias have kindly agreed to discharge that function.

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[1] The proceedings appear in *Life at High Altitudes*, PAHO Scientific Publication 140.

7

THE ORIGIN AND DISPERSION OF AMERICAN INDIANS IN NORTH AMERICA

James B. Griffin

The historically effective discovery of the New World by Europeans in 1492 opened the Americas to large-scale colonization and to a long period of speculation on the ultimate origin of the New World peoples. For almost four hundred years the chronological framework was based on biblical sources and was not adequate to explain the cultural diversity of New World culture. Prominent among the speculations on origins were the multiple-migration hypotheses, which provided civilized Mediterranean, European, and even Southeast Asian groups to bring a level of higher culture into the Americas and produce the more complex civilizations of Mesoamerica and the Andes. Such explanations were also employed to explain a presumed multiracial origin of the American Indian.

As early as the sixteenth century some writers recognized the predominantly Asiatic relationships of the native American populations and postulated that the movement of people took place from northeast Asia to northwestern North America. This is, of course, the only route of entry seriously considered by contemporary scholars, but there is considerable room for disagreement in many phases of the origin and dispersion of American Indian populations. For this paper I have chosen to refrain from presenting many reference citations to support the points of view included in the paper and from directly citing other views. Instead I have included in the references a list of books and other articles that afford a wide spectrum of interpretations and can be read by those who care to do so.

The temporal framework for early Indian groups

The time of arrival of the first human groups is far from settled and is likely to remain a research problem for some time. For purposes of this paper I shall refer to the period of time from the first arrival of man to about 8000 B.C. as the Paleo-Indian period, and all cultural complexes and skeletal material of this age, if any, will be included within this period. A rapid review of evidence from the United States and Canada includes only one fairly homogeneous and widely dispersed complex, a few examples of other early Paleo-Indian period peoples, and a number of purported sites that are not regarded in this paper as soundly established.

The Fluted Point Hunters are the sole recognizable group with a continent-wide occupancy in the latter part of the Paleo-Indian period from about 10,000 to 8000 B.C. The earliest radiocarbon dates in association with an established cultural context are about 9300 to 9000 B.C., from sites in Arizona, New Mexico, Colorado, and Oklahoma. This is referred to as the Llano complex, with the Clovis fluted point as a major diagnostic tool. The Sandia finds are regarded as a potential predecessor, with an uncertain age —probably within a thousand years of Clovis. In the western plains the later Folsom assemblages at several sites have been dated between 9000 and 8000 B.C. In the eastern United States and Canada, fluted points are known from the South

Atlantic and Gulf coasts to the Great Lakes and Ontario and into New England, and as far northeast as Nova Scotia. At the Debert site in Nova Scotia, a series of radiocarbon dates suggest that man occupied the area between 9000 and 8500 B.C. Provisional correlations of the distribution of fluted points in the Great Lakes area have proposed an age of from 10,000 to 8000 B.C. for that area, and the evidence from the Southeast also suggests an antiquity for fluted points of about the same order.

In the area west of the Rocky Mountains there are a number of complexes that may begin before 8000 B.C., but as yet this has not been adequately demonstrated. Among these are the Desert Culture, Lake Mohave and the San Dieguito culture of southern California, and a number of sites and complexes from Oregon to British Columbia and Washington compressed by some archaeologists into an Old Cordilleran culture. In extreme northwest Canada, the British Mountain complex is assigned considerable antiquity, but its age, except on typological grounds, is not known, and no archaeological assemblage from Alaska is as old as 8000 B.C.

The oldest radiocarbon dates in association with human habitation in the United States are from Wilson Butte Cave in south-central Idaho, where from the lower zone of Stratum C there is a date of 12,550 ± 500 B.C. (M-1409) and from Stratum E a date of 13,050 ± 800 B.C. (M-1410). The context of the material in the cave indicates the presence of man with extinct fauna such as camel and horse and some Boreal-zone microfauna, but the few artifacts found are not particularly diagnostic (7).

Fluted points are known from Mexico and as far south as Guatemala and perhaps Costa Rica. In none of these instances, however, is there an associated industry. Most of the few fluted points are found in northern Mexico, where they are on the southern fringe of the Llano and Folsom concentrations in the Southwest and western Texas. There are no soundly established cultural assemblages in Mexico and Central America directly dated by radiocarbon before 8000 B.C.

There are a large number of locations in North America for which considerable antiquity has been claimed as places inhabited by early Indians. Even whole books have been published on non-sites. The reasons why it is now difficult or impossible to include such "finds" here varies from location to location; a detailed dissent is not within the scope of this paper. In this category I would include the claims for occupation at Tule Springs, Nevada, of the order of 20,000 to 30,000 years ago; on Santa Rosa Island, much before 8000 B.C.; at the Scripps Institute bluff at La Jolla, California, slightly over 20,000 B.C.; at Lewisville, Texas, more than 37,000 B.C.; at Sheguiandah, Manitoulin Island, Ontario, for a cultural complex much before 7000 B.C.; for a completely pebble-tool culture in northern Alabama of extreme antiquity; for a chopper/chopping-tool complex of an interglacial or interstadial period, or of a simple bone-tool tradition of any age; or for Pleistocene man in the Trenton, New Jersey, gravels.

In Mexico particularly and also in Central America, there are indications of the presence of man before 8000 B.C., but many of these identifications were made quite a number of years ago and suffer from a lack of sound dates or are isolated artifacts inadequate for the reconstruction of a cultural assemblage. Some of these finds from the Late Pleistocene Upper Becerra formation may well record human occupation, and continuing excavations in Mexico will eventually place the temporal position and industrial activities during the Paleo-Indian period on a firmer basis. At present investigations are being conducted in the Valsequillo gravels of Puebla and at Tlapacoya in the Valley of Mexico. As usual, there is some uncertainty about the temporal correlation of gravel deposits between one area and another, about radiocarbon dates of 35,000 to 24,000 years ago of high antiquity but not directly associated with adequate artifacts, or about the precise age of a cultural complex on a buried living surface.

While the South American evidence for early Indian occupations is to be evaluated by Professor J. M. Cruxent at this session, I shall include some observations on this area because of its importance in assessing the probable age of the first inhabitants of North America. On the north side of the Strait of Magellan, in Fells Cave, there is a date of 8760 B.C. (W-915) obtained by Junius Bird. From eastern Brazil, at Lagoa Santa, there is evidence of occupation around 8000 to 7000 B.C., and a sambaquis on the southeastern Brazilian coast has provided a date close to 6000 B.C. In northeastern Venezuela, at the Muaco site, there is a possible association of man and extinct fauna in the period from 14,500 to 12,000 B.C. In the northern Andean area, artifact complexes have been given ages in the neighborhood of 8000 B.C.

The South American data, with their wide geographic spread of early man around or before 9000 to 8000 B.C., imply the arrival of man on that continent substantially before the known dated complexes. Similarly, in Mexico and Central America the wide distribution of human occupations just before or after 8000 B.C. implies that the arrival of the first human groups was substantially before this date. If the proposed dates for man in the Valley of Puebla and the Valley of Mexico are confirmed to be between 40,000 and 24,000 years ago, there will be much work for archaeologists to do in the future to find substantiating evidence in the rest of the New World. The North American geographical spread of dated evidence and the considerable diversity of assemblages shortly after 8000 B.C. imply an antiquity of man in North America considerably greater than the known age of the Fluted Point Hunters or of the occupants of Wilson Butte Cave. In summary, an age of about 15,000 years for man in the New World is viewed as reasonable, with the possibility that it may be considerably greater.

The temporal framework for northeast Asia

Sound dating of the Late Pleistocene occupations of Siberia is just beginning; in fact, adequate investigation of ancient man in northeastern Siberia has only recently been initiated. Most of the sites with an age of more than four or five thousand years are along the southern borders of Siberia from Russia to the Japanese islands. A radiocarbon date of 12,800 ± 120 B.C. (GIN-97) has recently been obtained on fossil bone from the lower cultural level of Mal'ta near Irkutsk, often attributed to the older Upper Paleolithic of the Irkutsk area. A date of 18,950 ± 300 B.C. was obtained on charcoal from the lower cultural level of Afontova Gora II in the Upper Yenesei Valley near Krasnoyarsk (*14*). From Kamchatka there is a date of close to 18,000 B.C. on charcoal from the Uski I site. Other sites in southwest Siberia are presumed to date substantially earlier, and sites in Japan of an Upper Paleolithic cast date well back toward the 20,000-year range.

The southern Siberian complexes of about 30,000 to 12,000 B.C. have a strong early relationship to the late Mousterian stone industries of eastern Europe. As the Upper Paleolithic developed, there was corresponding modification in Siberia, but the patterns of change in Siberia are sufficiently different from those of the better-known areas to the west that an easy alignment has not been possible. The industrial development of these Siberian populations was the result of the long Eurasian cultural development, which became adapted to the late Pleistocene. The level of occupation is attributed to the lower section of deposits on Terrace II of the Yenesei. The faunal composition represents cold periglacial conditions, and the date corresponds to about the maximum of the last major Siberian glaciation, called the Sartan. Also in the Middle Yenesei, at the Kokorevo sites, radiocarbon dates range from about 14,000 to 11,000 B.C. In the same area at the Mal'ta site, a date of about 7000 B.C. was obtained on a Mesolithic-like complex located on the higher areas of the Terrace I floodplain deposits.

In these Siberian sites there are crude heavy chopping tools, a variety of flake implements including scrapers and knives, discoidal cores, and some bifacially flaked points or knives. There is a trend toward greater use of true blades made from prepared cores and toward the fashioning of end and side scrapers, piecers, perforators, gravers and burins, and an increase in bone tools and ornaments. The animals on which the people fed are those from arctic to subarctic and cold arid steppe environments. They made skin clothing and had substantial houses in the construction of which they used the bones of large mammals such as woolly rhinocerus and mammoth. Probably the most important animal was the reindeer in the tundra area. It might be said that the spread of man into North America awaited the presence of arctic-alpine tundra species, during the late Pleistocene, on which man could live as he hunted his way across northeastern Siberia into North America.

While the extent of the mountain glaciers in eastern Siberia is not satisfactorily known, it is certain that only a small part of the land mass was glaciated and that most of the area was occupied by xerophytic arctic tundra or alpine tundra. A long tongue of steppe or periglacial steppe extended from southwest Siberia eastward between the Central Siberian Plateau and Lake Baikal as far as Yakutsk on the Middle Lena. From this area the best access route to the north was down the Lena Valley to the Arctic Ocean.

The fall of the sea level during the last major glacial advance of the Wisconsin-Wurm is estimated to have produced a land bridge at the Bering Strait from about 24,000 to 8000 B.C., with two periods of submersion of the highest part of the shelf corresponding to major ice-melting phases of the retreat of the Wisconsin ice. The size of the exposed land was considerable. Most of it was not forested but was occupied by tundra vegetation similar to that of the Siberian arid-steppe tundra. During the last glacial dominance, between 23,000 and 10,000 B.C., the arctic trees and shrubs were more restricted in their distribution than they are today and the climate was colder than it is now.

The movement of early man into North America

Keeping in mind the lack of direct evidence for the presence of early man in northwestern North America and northeastern Siberia, we can still present an acceptable hypothesis for a spread of hunting bands from west to east. Their way of life was developed from southern Russia to south-central Siberia during the latter part of the Pleistocene; it was based on a late Mousterian industry, modified by the initial elements of Upper Paleolithic emphasis on blade tools and the beginnings of a bone industry that was an aid in the production of skin clothing and shelter. This gradual expansion northward into new territories from northeastern Siberia to Alaska would have taken place without resistance from resident hunters. If it took place in the time period suggested, then a substantial number of hunting camps must now be under ocean water, but some will eventually be found in favorable areas such as elevations overlooking passes followed by game animals in moving from one feeding ground to another.

This early population spread is believed to have been diverted south along the west side of the McKenzie Valley. A number of recent papers (*9, 11, 28*) have emphasized the difficulty of passing from the Lower McKenzie Valley into the eastern Rocky Mountain slopes of the United States because of the presence of the coalesced continental and cordilleran ice from Montana to the Yukon Territory along the eastern margin of the Canadian Rockies. The evidence for the closing and opening of the corridor between these sheets is not so firmly established that sound datings for these events are available. The position adopted in this paper is that the corridor would have been closed only at the maximum of the Wisconsin glaciation for a few thousand years, about 19,000 to 15,000 years ago.

If the early hunters came into the United States before 17,000 B.C., then archaeologists in the United States either have been unlucky or have not been able to correctly evaluate the evidence for his occupancy before the 13,000 B.C. date mentioned at Wilson Butte Cave. Entry shortly before 17,000 B.C. would allow ample time for penetration into extreme southern South America for the known occupancy there, but would not accommodate the proposed Valsequillo and Tlapacoya occupations in Central Mexico. If the corridor was closed between

21,000 and 10,000 B.C., archaeologists are faced with at least as impressive dilemmas in the form of an absence of sound data representing man in North America before 21,000 B.C. and the long period from then to 13,000 B.C., or in accepting the speed with which man moved from Alberta to Tierra del Fuego.

The environmental changes in North America as a result of the retreat of the Wisconsin ice would have had an effect upon the way of life of ancient man through the shift of climatic zones, vegetation, and animal life. The expansions of the Canadian continental ice sheets effectively obliterated the vegetation and animal life from much of Canada. The expansion of the ice into the northern sections of the United States markedly altered the biota, and compressed and interdigitated elements of previous periods into assemblages distinctive to late Wisconsin times. The climatic conditions during the life of the western mountain glaciers lowered the tree line, changed the faunal associations and distributions, and produced thousands of lakes in the now-dry basins of the western Plains, in the Southwest, in the intermontane plateau region, and in the Pacific Coast states and Mexico. The lowered forest zones and more extensive and effective grasslands supported the large grazing and browsing animals of the late Pleistocene fauna. There were more streams, with corridors of pine and spruce crossing the grassland.

The changes in climatic regime accompanying the withdrawal of the Wisconsin ice had already produced notable shifts from the full glacial environments by the time of the early Fluted Point Hunters of 10,000 to 9000 B.C. The shift in vegetation and accompanying animal life took place on a large scale over North America, causing some shifts in hunting and collecting areas, and assisted in the displacement or disappearance of a small number of game animals. The northward movement of musk ox and mammoth is thought to have been in a park-tundra vegetation zone which initially occupied the soils left free of glacial ice in the Great Lakes area. It has been suggested that there was an early post-Valders invasion of the east by animal forms now associated with western prairie environments, and this would correlate with recent similar hypotheses of prairie vegetation movement eastward at an early period (*8*). The park-tundra and cool prairie would be suitable for barren-ground caribou, which have been identified in Michigan and New York. Did they penetrate this far south before the last Wisconsin advance, or did they arrive with the "reopening" of the corridor?

The Fluted Point Hunters of North America

By 10,000 to 8000 B.C. the people of the Late Paleo-Indian period had occupied sparsely most of the area south of the present Canadian boreal forest to South America and from the Pacific to the Atlantic. Most of these populations were strongly dependent on hunting, as we know from the spear and dart points, knives, and scrapers of various kinds to work skins and from the fact that these tools have been found in association with a small number of large game animals. This latter fact gave rise for a time to the idea of almost a limitation of diet to big game animals. Data from sites such as Lindenmeier in Colorado, Graham Cave in central Missouri, and others prove that the meat diet was quite varied, and at least in the east there is very little evidence of early man killing the mammoth and mastodon. In addition, the early Fluted Point Hunters would have recognized a large variety of the plant foods available, from nuts to berries and tubers. The diet of early man was not likely to stay restricted to a few classes of foods when he entered environments with a wide variety of them. It should be possible to discover sites that will reflect varieties of food gathering and processing activities that were part of the life of the Fluted Point Hunters. They should have had some seasonal activity patterns. A number of students of the Paleo-Indian cultures are beginning to recognize regional tool and behavior complexes that will aid our understanding of this earliest known complex.

The wide distribution of Fluted Point bands and the relative homogeneity of the implements recovered implies a rather rapid spread of these early hunting people, and apparently into areas not hitherto occupied. There is also the implication that there would have been continuing contact between neighboring bands, perhaps for group hunting or other food-procuring activities at favorable seasonal locations or at locations favorable for shelter during the winter seasons. Such collective activity would have permitted exchanges of new cultural developments—in terms of sources of food, raw materials, manufacturing techniques, hunting technology—and of people. It is doubtful that individual bands would have been isolated from other groups for extensive periods or that peoples moving into new regional environments would have been cut off by those environments from culture-sharing with peoples in their former territory.

Archaic-period adaptations in North America

In the long Archaic period in the United States between 8000 B.C. and the effective introduction of agriculture around 1 B.C., many regional cultural developments occurred as the Indian groups became more familiar with local resources and developed the knowledge for successfully exploiting them. As they did so, successful adaptations to particular environments tended to restrict band and group activity to these environments and to produce a higher level of exchange of culture and people within these areas than between them. This is reflected archaeologically by the growth of distinguishable regional cultural traditions.

One of the best-documented cultural continuities from the Paleo-Indian period Fluted Point Hunters to later complexes is in the western Plains states. The production of fluted points was gradually abandoned and nonfluted points and knives of essentially the same basic form continued in use along with the rest of the stone tools. New tools appear, such as the specialized Cody knife, and new techniques such as the parallel flaking of the Scottsbluff and Eden forms. All the evidence from sites in this region, from the Rio Grande north into the Canadian prairie provinces, continues to reflect the existence of a hunting economy with bison as an important supplier of food, tools, and clothes. At some locations there is clear evidence of mass killings, evidently the result of communal drives. Indications of variability of animal food comes from sites where giant beaver, pronghorn antelope, elk, deer, raccoon, coyote, and smaller mammals as well as bison were part of the food supply. At other sites there are indications of grinding and milling stones, burins, sandstone abraders, and whetstones.

West of the Rocky Mountains from around 8000 B.C., and continuing for many millenia, archaeologists recognize the Desert Culture, which has a number of named regional variants from Mexico into Oregon and Washington. These variants emphasize the gathering and preparation of small seeds by hand and milling stones; the hunting of a wide variety of animals; and the extensive utilization of wood, bone, hide, and vegetational sources for tools, ornaments, and containers. It was a gradually developing adjustment to the essentially desert environment, which supported only small bands and in which population density remained low up to the historic period. It was not, however, a static complex, for many significant changes took place in the technology, some of them representing almost continent-wide shifts in tool forms, and new weapons, and shifts in the techniques of manufacturing baskets. Important variants are recognized in areas along streams and lakes, in upland forested or alpine areas, and where minor shifting climatic patterns allowed the expansion of desert bands into sometimes better watered areas or the penetration of foreign groups into the Desert Culture region.

Between 8000 and 6000 B.C. along the Northwest Coast and into the interior along the major rivers, at least some part of the year was spent in obtaining food from the spawning runs and in otherwise exploiting the food supply associated with the streams and coastal areas. The latter is a reasonable inference, for some parts of what was then the coast are now under water. It was to be a long time, however, before the striking Northwest Coast sea-adapted complex would develop between Washington and the Alaskan peninsula.

In the interior, from the Yukon territory to Idaho, there is an Old Cordilleran complex that may be viewed as an Asiatic-derived parent to the Fluted Point, as a collateral contemporaneous variant, or as the result of the northern and western expansion of the Llano to Plano tradition. At present there would seem to be a basic relationship, and current radiocarbon dates indicate the time period of Old Cordilleran as not over 8000 B.C.

In southern California the San Dieguito hunting culture with percussion-flaked lanceolate points, knives, scrapers, and choppers is known from about 8000 B.C. to 6000 B.C. Shortly afterward there is a development of a number of areal specializations in coastal, desert, and forest environments, which during the last few thousand years resulted in an unusually dense population for a hunting-gathering population in the oak-forest area of central California.

Southwestern Alaska was sparsely populated by around 8000 to 6000 B.C. by people with a unifacial core and blade industry whose movement into Alaska is likely to have been from the Pacific side of the Chuckchi peninsula into coastal Alaska and south to the eastern Aleutian area. If these early coastal-adapted groups were the first Aleuts, as is implied, it would suggest that the Bering Strait area was occupied by Eskimoan-speaking peoples longer ago than has been thought. In interior and northern Alaska there is a considerable variety of assemblages reflecting inland and coastal developments with continuing ties with Siberia, and also influences from northward-spreading groups primarily moving with the expansion of bison and other game animals.

Between 3000 and 2000 B.C. the Denbigh Flint Complex, with a marked coastal adaptation, spread with surprising speed eastward to provide the first successful occupation of the eastern Arctic. The first Eskimo bands reached northern Greenland by 2000 B.C. Eskimo cultural traditions have a considerable time depth, and a main hearth area was the Bering Strait on both sides of the International Date Line, where people have been moving across in both directions for many millenia.

In the woodland area of the eastern United States a gradual transition is recognized in several areas from the Fluted Point Hunters complex to assemblages maintaining the same basic manufacturing stone-working techniques and tools, but with the development or adoption of nonfluted projectile forms very similar to some of the early Plano forms of the Plains. By 7000 to 6000 B.C. stemmed and notched projectile forms become common, and an increasing diversity of regional areas through time reflects the increasing specialization of groups learning to exploit the resources of these local regions. Many of the changes are related in form and function to those of areas to the west, but the east acquires a distinctive flavor of its own through the development of a series of woodworking tools and ground and polished stone forms. While regional specializations are present in the eastern Archaic complexes, there is also evidence of increasing exchange of raw materials or manufactured objects as travel or trade routes become established.

Many archaeologists view most of the cultural complexes of Mexico from about 8000 to 4000 B.C. as a southern extension of the Desert Culture. Certainly the general hunting-gathering pattern is similar, and there are some tool forms that are held in common with early Archaic and Desert Culture groups from California to Texas. The most important feature of the Mesoamerican scene is the early domestication of plants, from Tamaulipas to Chiapas, which has been demonstrated where systematic efforts have been expended to search for such evidence. This area shows a very gradual increase in the number of plants domesticated in the several regions and in the proportion of domesticates consumed. Marked population increase in some areas such as the Valley of Mexico, the Valley of Oaxaca, the coastal lowland of southern Vera Cruz and Tabasco, and the Pacific Coast of adjacent Chiapas and Guatemala is observed by 1000 B.C., when agricultural practices were well developed and the Early Formative cultures were becoming established.

Summary

All the supportable evidence available indicates that the first human occupants of North America came from northeastern Asia. Some archaeologists support the view that this first occurred from 30,000 to 40,000 or more years ago, others believe it was from about 25,000 to 20,000 years ago, and some have contended that it could not have been until about 12,000 years ago. The time of arrival has not been settled.

Some archaeologists emphasize the Mousterian origins of the first emigrants, believing that the earliest American cultural complexes indicate a spread into North America before elements of Upper Paleolithic origin had reached eastern Asia. Many archaeologists, however, believe that Upper Paleolithic developments were a part of the cultural mechanisms that allowed man to move into North America and spread throughout the New World.

The main access route into interior North America was east of the Rocky Mountains, and dispersion into most of the Americas was by this route. Population increase and any physical differentiation of human groups south of the Arctic area is derived primarily from the populations of the Paleo-Indian period. There are no indications in the prehistoric record of any later substantial migrating groups influencing the cultural life of the residents of North America south of the Alaskan and Canadian Arctic region.

The archaeological evidence, except in rare instances, supports the view that, in spite of regional adaptations to food supplies and raw materials, there was a continuous exchange of new developments between regions, with the additional implication of population interaction as well.

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THESES FOR MEDITATION ON THE ORIGIN AND DISTRIBUTION OF MAN IN SOUTH AMERICA

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This paper does not constitute an extensive study of the origin and early settlement of South America. So many important works on the subject have been published that I am relieved of that duty. I need only cite Hester (*10*), Krieger (*12*), Comas (*3*), Lanning and Patterson (*13*), Lynch (*14*), and Bosch Gimpera (*8*).

Unfortunately, despite all the valuable recent documentation, we still cannot really prove just how South America was peopled.

As we all know, concern over the problem of the origin of American man goes back to the time of Columbus. The conquistadors even wondered whether Indians were human, and so it is understandable that Pope Paul III should have issued a papal bull explaining that the Indians were in fact human and could receive the Faith (*18*).

Since the sixteenth century numerous hypotheses about the origin of American man have been suggested, and most of them have been discarded as fantastic. Today we feel that we have long outlived that era, that our work is imbued with a scientific spirit and our hypotheses are based on valid archaeological documents. Nevertheless, I find it difficult to accept that *all* this vast ancient documentation is worthless. I say this in full awareness of how difficult it is for a present-day archaeologist to consult and study this copious literature, which not only is hard to find but is sometimes available only in archaic languages. But I wonder whether prejudging all those ancient studies without even knowing anything about them is a responsible thing to do. In many cases I think it may be precipitate. I am convinced that a review of the old literature in the light of modern research might be very useful and might lead to surprising conclusions.

Out of the great variety of working hypotheses on the origin and dispersion of man in South America, which I consider of little help to those concerned with human biology, I find one positive fact: even today we cannot be sure that man in South America came uniquely by land and by way of Panama.

After years of trying to find an answer in archaeology and other sciences, I believe that South America was peopled not only overland but also, and to a large extent, by sea and river routes, primitive though the means of navigation must have been. I do not see how men could otherwise have crossed so many rivers, swamps, and lakes with their entire families—women, children, and the aged—to occupy the whole of the South American continent.

If the route was terrestrial, the only entrance is through the Darien jungle in Panama and the Atrato region in Colombia. I know that area well; I have crossed it on foot from the Pacific to the Atlantic and back again. In this labyrinth there is no sun, only a weak green light. At every step is a river or a marsh. Whether through the low ground or along the mountain crests, walking is extremely difficult; hunting would have been practically impossible. In the "winter" or rainy season no one could have moved. And there are many other Dariens, and many rivers to cross. From my travels in South America I suspect that Paleo-Indian man must have had to go at least part of the way by water (and I make bold to suggest that he may have known how even before he entered the New World); if so, this would explain many of the gaps we find when we try to trace his migration. Unfortunately, a great deal of the possible evidence has disappeared under rising water levels since Paleo-Indian times (*10*).

The immigrants were unquestionably required to adapt themselves to a great variety of environments, but if they traveled by water the cultural changes would be less strong and violent than if they came only by land, because there would be fewer stages to their journey. Genetically, they might have had fewer contacts or mixtures with other migratory groups because the continent was so sparsely populated at the time. But if we accept the possibility of a water route, we must also accept the possibility that some groups reached South America some other way than through Darien.

The "autochthonous" theory of American man, which originated in a misreading of Quaternary fossil remains and in the belief that Paleo-Indian immigration would have been impossibly difficult, seems now to have been rejected—among other reasons because there is no convincing evidence to support it. I should like to make the point that a good way of laying it to rest once and for all would be to prove that man came from elsewhere and to prove how he came. I may say in this respect that, even though I cannot prove the contrary, I am not at all satisfied with the Bering Strait as the sole entrance route. How could man have spread over such a vast territory and created such a mosaic of cultures and ethnic groups in so short a time, 20,000 years at most, if he had entered by a single route?

I have the impression that not enough attention has been paid to the ancient accounts of the travels of such navigators as the Phoenicians, the Romans, the Arabs, the Vikings, and the Normans. Apart from the reasons I mentioned above, there is the general discredit to which these theories are subject because they have been put forward by untrained persons guided by ignorance or by a desire to sell books. The scorn of responsible archaeologists, and their unwillingness to risk their prestige by examining them critically, is understandable. But there are some recent finds that if verified would be of great interest. I do not believe that the seriousness of such studies as those of Hui-lin Li (*11*) or Sauer (*22*) can be doubted.

One of the old chronicles tells of finding Old World coins in America, and I myself know of a North American who bought a pot full of Roman coins, which seems to have been found in a cave, from a farm worker in Falcón State, Venezuela. The coins are now in the Smithsonian Institution. I have also seen in the Jamaica Museum a pedestal with a Roman inscription that was found off the Jamaican coast. I have heard that a Mexican publication, which I have not seen, mentions a small Roman statue unearthed in that country. The newspapers are forever reporting discoveries of ancient objects of European and African origin—discoveries, however, that are seldom controlled or studied by professional archaeologists. I bring all this up merely to inquire whether it is proper for us to consider such information *a priori* false and of no interest.

Many colleagues have asked me: "If these arrivals did occur, where is the evidence? Where does archaeology find any appreciable traces in the indigenous cultures?" This is the problem to which we must find an answer, not only from the cultural standpoint but also from the biological.

As to the former, let me say that the cultural impact of a small immigrant group on an American aboriginal population is in most cases practically nil. We have an example in Balboa's siege of Careta: ". . . the chroniclers agree . . . that Balboa found helpful compatriots already installed there. . . . They had adapted themselves to their new environment without reserve, and . . . all were 'as naked as the Indians and as plump as the capons that housewives fatten in their cellars'" (*20*). This probably happened on many other occasions, and not only at the time of the Conquest. When I was exploring the Orinoco Delta region I came upon a Frenchman (he eventually confessed that he had escaped from Devil's Island), living with an Indian wife and mestizo children; it was only by his slightly lighter skin and his beard that I was able to identify him. I have heard of other examples, and there must be many more.

As can be seen, the cultural influence of the intruder is slight or nonexistent. His biological influence I cannot say—that is up to the biologists.

The possibility of immigration by sea is supported by the fact that navigation is much easier than is supposed and much older than is generally accepted. In the Old World, from prehistoric times until the fifteenth century, boats hugged the coasts, for the fear of the open sea was great and navigation was rudimentary. But any storm might pull the boat out to sea, and then the crew had to resign itself to the currents and the wind. When it is recalled that some of the remote Pacific islands were peopled by the time the Europeans came, the logical conclusion that man willingly or unwillingly made long journeys across the oceans cannot be avoided. The most fortunate encountered islands or continents; some learned in time how to get back again, and others did not.

Columbus' diaries, citing encounters with canoes that traveled the Caribbean from island to island, support the idea that sea journeys are possible even in small, primitive boats. We also have the many incredible transatlantic voyages of sportsmen (*16*) and contemporary political refugees who have brought small, crammed boats across the ocean in search of freedom and a new life in America. I do not believe that Bombard (*1*) was the first to obtain potable water from fish; this vital technique may well have been known to the ancients. In preceramic times they could have stored it in light-weight vessels such as gourds or skins, which when empty could have been used as floaters.

Once again, therefore, I postulate navigation as a possibility that could have spared the Paleo-Indian innumerable obstacles on his way and that furthermore would have permitted migratory movements without requiring the existence of an ice-free corridor east of the Rocky Mountains.

Origin and antiquity of man in South America

All recent studies on the origin of man in South America agree that migration was channeled from north to south across the Isthmus of Panama. Evidence of this migration is limited, perhaps because there has been very little exploration in this area. So far, no other theory seems to have been confirmed, not even that of Mendez Correa (*15*), who suggests a route through the Antarctic from Australia to Tierra del Fuego. As far as I know, no transatlantic routes have been proposed. However, extraordinary similarities have been shown between the stone implements of western Africa and northeastern South America (*5*). These parallels are much closer than between Paleo-Indian material from Siberia and from North America.

As to the antiquity of Paleo-Indian man—using this term for a hunting and gathering culture that coexisted with animals now extinct and is known by its crude chopping tools —no accepted chronology yet exists. We may estimate that the Paleo-Indian disappeared about 7,000 years ago, and very tentatively we may propose an arrival date in South America or Venezuela of 15,000 to 20,000 years ago. This latter date is calculated on the basis of finds for which the following ages have been obtained: El Jobo complex, 10,000 years; Las Lagunas and El Camare, more than 16,000 years; and Muaco, 14,740 to 16,580 years (*21*). I do not know of any earlier dates anywhere else in South America. Lanning and Patterson (*13*) estimate the Chuqui complex in Chile and the Tortuga and Red Zone complexes in Peru as the most ancient in the Pacific Andean region, both being 13,000 to 14,000 years old. For Argentina we have a date of 7,970 ± 100 at Ayampitin-Intihuasi, and for Brazil a date of 7,500 at

Sambaqui de Maratua (*18*). Current excavations in Chile showing man in association with mastodons and a variety of other animals promise to produce extremely important documentation (*2*).

From these and a few other dates that might be mentioned, we can make the following tentative timetable for Paleo-Indian man in America: North America, 15,000 to 30,000 years (*9*); northern South America, 10,000 to 20,000 years; western and southern South America, 8,000 to 14,000 years. The figure for North America deserves a question mark; the experience of South America leads one to believe that further studies will uncover earlier dates (*13*). Indeed, although it is quite possible that some specialists may consider these dates too ancient, I find them not only logical but overly conservative and I expect future studies to at least confirm them.

Principal lithic complexes

Lanning and Patterson (*13*) have proposed a three-group typology for the South American lithic complexes: (1) bifacial industries, (2) burin or graver industries, and (3) chopper industries. An examination of the principal Venezuelan complexes shows that the typology of that country presents certain characteristics that are not present in those of Argentina, Chile, Peru, and Ecuador. Earlier this year, when he consulted our collections in the Venezuelan Institute for Scientific Research, Dr. Lanning was able to relate the Camare complex with the "Bifacial Andean Horizon" (large chipped artifacts, large bifacial stones, absence of projectile points). He could not relate the Chuqui, Tortuga, Red Zone, or Exacto (Ecuador) complexes with those of northeastern South America, and he found some of the Lagunas and El Jobo material (plano-convex scrapers, spear points, and other articles) to be quite unlike anything in the regions he had studied.

To me this circumstance is extremely important. It permits a working hypothesis as to the existence of another possible entrance route to South America: the chain of Caribbean islands from Florida to northeastern South America, which I accept for both Meso-Indian and Neo-Indian times. I may cite here—purely for reference purposes, since I am not sure of their validity for the Caribbean—Hester's observations (*10*) on the rising of the sea level since Paleo-Indian times. Many islands would be interconnected, thus making navigation and migration easier. The C-14 ages of some Meso-Indian sites support the idea of inter-Antillean navigation: Rooi-Rincon, Curaçao, 4490 ± 60 years; Mordan, Dominican Republic, 4120 ± 160 years; Punta Gorda, Venezuela, 4150 ± 80 years.

As to Paleo-Indian man in the Caribbean, at the Mordan site we have found a more ancient level of chipped-flint implements, which we have called Alejandrina (Cruxent and Chanlatte, work in progress). The typology of the Alejandrina complex can be related to that of the El Camare complex, with large plano-convex knives, scrapers, chisels, and other tools. This material has not yet been fully studied. The artifacts are made of excellent-quality flint and are in strict accord with the typology of flint industries, which is based in part on the nature of the substance. The same is true of the Manzanillo and El Camare implements, the first of which are of silicified wood and the second of quartzite. I am one of those who believe that a single group of artisans trying to manufacture a certain type of instrument with different materials would have to produce different typologies. This must be taken into account in comparing complexes, for it is easy to make mistakes. There is also the possibility of the fortuitous use of objects as implements in certain circumstances. This may be the answer in the case of Taima-Taima, which is completely atypical; on the other hand, it is just as likely that we call it atypical because this is the first time we have seen such implements in association with Pleistocene fauna.

The presence of Paleo-Indian culture on the strategic island of Hispaniola raises a series of problems. The principal one is how the Alejandrina people lived, for no fossil remains of mastodons, glyptodonts, or megatheres have been found. Considering that they were islanddwellers, we must conclude, if they were great hunters, that if large animals were not to be found on land they may well have been found at sea—perhaps manatee and seals, both of which were abundant in the Caribbean in those times (*7, 23*).

Nothing is known of the origin of the Paleo-Indians of the Alejandrina complex, but they probably came from the north. Nor is it known whether they reached Venezuela, though some slight contact between the Alejandrina complex, or an older one of the same sort, and the El Camare and Las Lagunas complexes would not have been impossible. Later groups may also have had some influence on the early Meso-Indian or late Paleo-Indian complex of Canaima in Venezuelan Guiana (*21*), the Rio Claro complex in Brazil (*4*), and the Gran Sabana and Paragua material.

I hope in the near future to be able to clear up this problem of another route to eastern South America in Paleo-Indian times. This would extend the validity of the H theory proposed by Osgood and Howard (*17*) as a graphic explanation of migratory routes in general.

Before concluding, I wish to point out that many times we fail to take into account a hypothesis that should not be ignored in drawing conclusions. I am referring here to the possibility that there may have been Paleo-Indian groups without a lithic industry. I do not understand why we always imagine Paleo-Indian man with a stone implement in his hand; we are even presumptuous enough to demand a specific type of implement, an officially accepted one. We must not forget that until very recently projectile points were the only implements recognized; all others were considered "blanks." For many years I have been accumulating experience with an archaeologist's eye in many Indian communities. This *in vivo* archaeology has given me many answers that would have been hard to come by from purely academic studies. (Of course, this often puts me in the unfortunate position of having no erudite citations to make in support of my arguments.) From this experience, I do not think we can discard the possibility that there were Paleo-Indian groups without stone implements any more than we can ignore the likelihood that they knew how to navigate.

I also think it a valid working hypothesis that Paleo-Indians without a lithic culture, which I call "primary culture groups," could well have been the ancestors of certain groups of forest gatherers and primitive agriculturists in South America. Lithic culture evolved through a series of phases before arriving at triangular pedunculated points; it is significant that this tradition survives to the present day in our *llanos* and savannas but that I have not found it in jungle or thickly forested areas.

This could indicate that a diversity of diets existed from the start or else that it came about at the end of Paleo-Indian and the beginning of Meso-Indian times, when the great vertebrates disappeared. Unfortunately, no conclusion can be reached at present, for we do not know whether the jungle-dwellers descended from another group of great hunters or from a group that entered the New World already carrying a primary culture. It is also possible that some groups abandoned the flake industry in adaptation to an ecological area that did not allow its use. Geography is not determining, but it undoubtedly affects many aspects of the life of primitive peoples, who obtain from their habitat the elements essential to their needs and their security.

In conclusion, I believe that many of the gaps we observe in Paleo-Indian migration can be logically explained by taking navigation into account. I do not reject the idea of terrestrial migrations; I wish only to complement it. In any case, I am optimistic about the possibility of solving the problem of the origin and dispersion of man in South America, because most good archaeologists do not regard it as settled.

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DISCUSSION

William S. Laughlin

I feel a little like an Eskimo shaman coming before the College of Cardinals to discuss the doctrine of the Immaculate Conception. But an Eskimo shaman would not hesitate to do this, and so neither will I.

A great deal of ground has been covered very expertly by both these scholars, and they have raised a number of very good problems. I can make only a few comments and perhaps add one or two observations by way of interpretation.

I would suggest that most or all of the people who entered the New World came by way of the Bering land bridge. This, of course, does not exclude other points of entry. But at least the other groups that may have come in have left no distinguishable genetic heritage that can be sorted out.

As Professor Griffin pointed out, the Bering land bridge was a large extent, over 1,000 kilometers from north to south. This leaves, then, an opportunity for at least two routes across the land bridge and may help to explain some of the differentiation between the less Mongoloid American Indians and the more Mongoloid Eskimos and Aleuts of the North.

It is very likely that some of the early people lived permanently on the land bridge. They were there for a long period of time. They did not simply run across in order to get to the other side in order to be discovered by people who came later. And for all practical purposes they were unaware that they were going to be submerged. So for a few thousand years they enjoyed the real estate they held.

Now, those who lived on the coast had, I think, the advantage of a richer ecological zone. They had the marine mammals. They had the fish. And, of course, they had the birds, as well as those things that grow in the intertidal zone, and, very importantly, invertebrates, mollusks, octopus, and so forth. these last are foods easily accessible to women and children.

I suspect that the ancestors of the Aleuts (of the Aleutian Islands) and the Eskimos (of the mainland extending over to Greenland) may have originated or taken their primary form on the southern part of the Bering land bridge, and that when the land became submerged they simply backed up and remained on the coast. Of course, as the waters rose there was more coastline rather than less, and therefore they prospered rather than disappeared.

The ancestors of the Paleo-Indians, who more likely came through the interior, may have never seen the people on the coast, being separated by several hundreds of miles, or have had only intermittent contacts with them. They were big-game hunters hunting mammoth, caribou, and possibly musk-ox. These people may have lived on the Bering land bridge for some time, but when the waters rose they had only two options. One was to learn to live under water; the other was to go back to Siberia or on to the New World. Obviously, a number of them went on to the New World.

Now, when we look at the early sites, we find very good documentation for the period between 9,000 and 12,000 years ago. There are a great many dates that I think can be accepted as being well replicated. But getting beyond the 12,000-year barrier is difficult.

The most recent piece of evidence is one that Dr. T. Dale Stewart called to my attention yesterday. Dr. Leakey has rediscovered an old discovered skull in Southern California, at Laguna Beach, which has been dated on the bone itself—not on associated materials—at 17,000 years. The skull has not arrived here yet, and we do not know what it looks like.

In any event, if that date should be substantiated, we would be in a position to say that man had to come prior to the 14,000- or 15,000-year period and must therefore have come before 24,000 years ago. This, however, has not yet been established.

Now, turning to South America, one thing I should like to mention is the trait complex associated with the Lagôa Santa crania of Brazil. You will recall that these were discovered in the last century; they are now in the Museum of Zoology in Copenhagen.

Lagôa Santa crania are dated around 8760 B.C. The interesting thing is that although they appear to be this old, they look like those of contemporary Indians, and not only in general features of morphology but also in discontinuous traits that may be more similar to single-gene traits, though this is not firmly demonstrated. They have shovel-shaped incisors, which are found in all New World occupants, both Eskimo and Indian. They have dehiscences of the tympanic plate. And there are several cases of interparietal bones, the separate Inca bone on the posterior of the skull, which reaches a high frequency in South American Indians.

Taking this example to raise a point, it would appear that there has been very little morphological change in American Indians over a period of 9,000 years.

We can say the same thing for North American, though we do not have as good a series as for Lagôa Santa. Nevertheless, the amount of morphological evolution does not appear to have been great.

We can go one step further and point out that the Eskimos and Aleuts, who form a single coherent population system, demonstrate a similarity to Middle Pleistocene man of northern China, as was originally observed by Franz Weidenreich. This similarity probably indicates some genetic continuity. If that is so, this again reflects a rather minor amount of external visible morphological change and suggests a slow rate of morphological evolution.

One interesting point we must consider then is the possibility that the morphological divergence between peoples within the New World and between the inhabitants of the New World and the Old may reflect a slow rate of human evolution, morphologically speaking. Therefore, the fact that some of the earliest physical remains do not look like Neanderthal man or like Australians does not mean that they have not been here for some time.

And there is something else to be emphasized—that the amount of morphological change does not accurately reflect that of other tissue systems. Serological evolution, dental evolution, changes in different tissue systems, are proceeding at different rates. When we look to the past, we are limited to the skeleton; we cannot assume that change in many genetic traits, including behavioral traits, has not gone on at perhaps a much faster rate.

The other point I should like to make is the fact that the majority of the people coming across Bering Strait were exposed to a cold filter. All botanists, glaciologists, geologists, and anthropologists are agreed that, whatever conditions were, they were cold. The inhabitants adapted to the cold for the few or several thousand years they lived on the Bering land bridge. This may possibly be related to some of the epidemiological and immunological aspects of contemporary populations. Once they got south, of course, they could adapt to several other varieties of zones. But they all had to pass through a cold filter.

The last point I should like to mention is the rate of dispersion and how long it would have taken to get to the Strait of Magellan. I simply call your attention to the fact that the Aleuts and Eskimos are spread over a difficult expanse of 8,000 miles, and it took them considerably less than 8,000 years—possibly as little as 2,000 years—to get that way. Therefore, the trip to South America may not have been as long as we might otherwise think.

General Discussion

Salzano: Since Dr. Laughlin mentioned the Lagôa Santa material, I want to say a few words about the prehistoric remains of Brazilian Indians.

A few years ago a researcher from the National Museum, Professor Marilia de M. Alvin, did a review of the anthropometric data at hand and found a considerable heterogeneity between the different Lagôa Santa sites. We cannot say, therefore, that these people are just one homogeneous group. She also examined the shell-mound material of different locations and they were even more heterogeneous.

Moderator: Do you mean to imply that the heterogeneity in the Lagôa Santa material is greater than might be found in contemporary Indian populations in Brazil?

Salzano: That is difficult to say, because the material available for study is not very extensive —only about 80-odd individuals. But even in this small sample the heterogeneity is very great. I am not saying, however, that it is greater than among present-day Indians.

Roche: I have a question for Professor Cruxent. He gives a possible age for man in North America of from 15,000 to 30,000 years, in western and southern South America of 8,000 to 14,000 years, and in northern South America of 10,000 to 20,000 years. These ages seem to go from more to less. Does he consider this as one more proof of north-to-south migration?

Cruxent: I do not think the last word has yet been said as to ages. We have not been able to date the oldest complexes we have in South America, in the alluvial terraces at great altitudes, because all the material is on the surface and has been exposed to the air. It happens in some places in the States. But I feel that little by little, here in the States, earlier ages will be found. We keep finding older and older material in America, and I think that will continue.

Roche: That was not exactly my question. What I asked is this: You seem to give the impression that in North America we find older ages than in Mesoamerica, and in turn the ages in Mesoamerica are greater than those in South America. Could this be taken as additional evidence for a north-to-south migration?

Cruxent: Yes, it seems to confirm the theory that the migratory current was from north to south.

Moderator: Professor Griffin, you have a name for being a bit of a skeptic in these matters. Would you care to respond at all to Dr. Cruxent's presentation?

Griffin: The evidence of man in South America, both what can be established and what has been claimed, indicates an antiquity of man in South America greater than that in North America, and evidence in which I have some confidence suggests that we have not found man in North America of the antiquity necessary for his appearance in South America at the age claimed. I think it is possible that we simply have not found the evidence in North America.

I should like to comment on one point of Cruxent's paper: If through man's penchant for self-destruction the evidence for our astronauts were completely destroyed, there would be very little in the earth's atmosphere to tell the people of the future that man had actually gone around the world. Cruxent's "Paleonauts," if I may coin a phrase, are extremely difficult to trace in the sea waters. So that while he hinted broadly at the possibility of man's making voyages from western Europe or western Africa and perhaps across the Pacific, my position is that he may have done so but I have seen no traces of the boats, or whatever they used, and thus have no evidence for such an interpretation.

I also wish to comment on the presumed contact of Mediterranean, European, African, or relatively civilized Asiatics with the New World. Such ideas have been current for a long time. They were originally based on the philosophical position that it would have been impossible for Indians to achieve high cultures without benefit from groups that had achieved reasonably high cultural status in the Old World. But that position has been abandoned. We no longer need to bring high culture into the New World from the Old because now we can clearly document the gradual development of the higher civilizations in the New World, both in South America and in Mesoamerica.

It is true that a great many contact areas are known. For example, there are stories of the Irish being present in North America, primarily around the hinterland of the Boston area. Phoenicians are recognized in eastern Pennsylvania around Lebanon. A major center for pre-Columbian Scandinavian influence in North America is in the area of Minnesota and Wisconsin. But some of us are extremely skeptical about the appearance of Roman coins or statues or various other objects, because they can be reasonably explained on the basis of man's pack-rat and souvenir complex.

And so I admit to somewhat of a skeptical position on many of these matters that Dr. Cruxent has so delightfully called to our attention.

McDermott: Dr. Griffin, is there any linguistic evidence acceptable to reasonable man, as you put it, on this question of migration from North to South America?

Griffin: I am told there was tremendous linguistic diversity among the American Indian groups and that none of the New World language stocks can be traced back to those of Asia with any degree of certainty. I am also told that the linguistic diversity in, for example, the eastern United States could reasonably have taken place within a period of some 10,000 to 12,000 years from a single ancient linguistic stock.

The archaeological evidence we have indicates to some of us that the original populations of the eastern United States were relatively homogeneous in cultural material and, further, that cultural extremes can best be explained primarily as regional developments based on the original cultural groups.

Waterlow: Dr. Laughlin suggested that two groups of people came over by different routes—northern and southern—through Alaska, one being the rather small Eskimo type and the other the North American type. Is there any evidence that they came from two different families or groups in central Asia?

Laughlin: I think the answer is no, there is no evidence of two separate groups that can be distinguished today. However, the record in north China and Japan does suggest that evolution has been proceeding there and that people would differ according to when they sampled that particular area.

So the fact that the more recent (if indeed they are more recent) Eskimos and Aleuts look more like Japanese and Chinese may reflect either their recency or the fact that they were drawn up the north Pacific rim—Japan, the Kuriles, Kamchatka—and followed the coast rather than coming from northern Siberia.

There is no particular group that could be identified today, and I think it would be hazardous to look for one, since those populations have been evolving for the same period of time as those in the New World.

But there is, as Dr. Comas and others have pointed out, a major dichotomy between the Aleuts and the Eskimos and the rest of the Indians. This includes several serological factors as well as growth rates, disease patterns, and other morphological differences.

I would add just one other thing, and that is that in our views we should take into account that Japanese anthropologists have again pointed out that they think the Ainu of Hokkaido, in northern Japan, are a variant of Mongoloids and not of archaic whites who were surrounded. The old thesis, originally proposed by Professor Birdsell, that there was a group of Australoids, archaic whites, or Ainu-like people living in Siberia is no longer tenable if as much heterogeneity as that between Ainu and Japanese can be recognized among Mongoloids.

Griffin: If our archaeological evidence is any good, then the first migrations seem to have been from the central Siberian area, with cultural complexes that date back somewhere around 20,000 years or so. These populations seem to have moved primarily along the northern coastal areas of Beringea into the North American interior. The evidence is that the populations Dr. Laughlin has been talking about with his proto-Eskimo or prehistoric Eskimo of 8,000 years ago or more are distinctly different from the earliest movers into the New World and had a cultural complex formulated primarily on the Pacific side of northern Asia, at a later time. It was chiefly the first group, moving into the New World along the northern rim, that populated North America and I think moved into South America, either on migratory-bird wings, across water by navigation, or plodding through and slowly adapting to various environments.

Roche: I want to make just a brief comment in relation to Dr. McDermott's question. There have been studies on linguistics, but there have been very few comparative studies on the music of the Indians. I have been told by the head of the Institute for Music Study in Berlin that primitive music shows much less variation and evolution than primitive languages, that origins can be recognized much better through music than through language. Practically nothing has been done in this area. For what it is worth, he feels that the Latin American music he has heard, particularly the very primitive, such as that of the Caribbean, has very strong relationships with northern Asia, particularly Japan.

Moderator: At this point we have succeeded in getting the Indian to the Americas, although there is something less than unanimity as to how this was accomplished. In the next section of the program we propose to look at the physical and genetic diversity present in the American Indian. The distinction between "physical" and "genetic" is of course artificial, since the basis of much physical diversity is genetic. It is a convenient distinction, however, and we shall adhere to it today.

BIOLOGICAL SUBDIVISIONS OF THE INDIAN ON THE BASIS OF PHYSICAL ANTHROPOLOGY

Juan Comas

In accepting the invitation to participate in this Special Session, I was guided primarily by the great interest this subject has always had for me. I well realize, however, that my knowledge of the problem and my information, both direct and indirect, leave much to be desired. I present my excuses in advance and ask for leniency on the part of my readers.

For a better understanding of the value and scope of the principal biological subdivisions of the Indian on the basis of physical anthropology, I believe it necessary to first pose three questions and go on from there to some provisional conclusions. The questions are these:

1. Do the American Indians constitute a biologically homogeneous population or have they, on the contrary, a certain variability which permits thinking in terms of subdivisions?
2. To what cause or causes may the biological variability of the American Indian be attributed?
3. What attempts have been made at a racial taxonomy of the American Indian?

Let us examine these questions consecutively.

Biological variability of American aborigines as against classical conception of "American homotype"

The excellent works of Newman (*47*) and Stewart and Newman (*56*) make it unnecessary to devote too much space to this question. Discrepancies on this point have a long history. Antonio de Ulloa's statement toward the end of the eighteenth century that "If we have seen one American, we may be said to have seen all, their color and make are so nearly alike," and the consequent acceptance of the somatic unity of the pre-Columbian population of the New World as a definitive fact, received the support of such renowned anthropologists as Samuel G. Morton (1842), Timothy Flint (1826), Ales Hrdlicka (1912), and Arthur Keith (1948).

The opposite camp—composed of those who recognized the existence of obvious biological differences among the Indian groups, describing them as "races," "varieties," or "sub-species"—included, among many others, Humboldt (1811), Desmoulins (1826), D'Orbigny (1839), Retzius (1842), Aitken Meigs (1866), Topinard (1878), Deniker (1889), Virchow (1890), Ten Kate (1892), Haddon (1909), Biasutti (1912), Wissler (1922) Dixon (1923), Rivet (1924), Eickstedt (1934), Hooton (1937), Imbelloni (1937–58), Count (1939), Neumann (1952), and Schwidetzky (1952).[1]

It is interesting to point out that, while the believers in the somatic unity of the Indian remained a minority, in their forefront was Morton, of whom Stewart and Newman (*56*) say shrewdly:

> Indeed, so great was his influence that he was responsible in large measure for the wide acceptance of the generalization embodied in Ulloa's words and for the conversion of Ulloa's words into an "adage." (p. 22)

Morton's influence lasted for more than half

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[1] Most of these are well-known general works and therefore do not appear in the References. For additional information see J. Comas, *Manual of Physical Anthropology*, (Springfield, 1960), pp. 81–86 and 588–600.

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a century, until Hrdlicka arose as the new champion of the homogeneity of the Amerindian. In summarizing his arguments Hrdlicka said in 1912 (*32*): "The conclusions are that the American natives represent in the main a single stem or strain of people, one *homotype*." He reaffirmed this thesis in 1928 (*33*) in an attempt to refute those who supported the racial plurality of the American Indian:

> We find that the various differences presented by the Indians are often more apparent than real; that actual and important differences are in no case of sufficient weight to permit of any radical dissociation on that basis. (p. 481)

This position had the able support of Sir Arthur Keith (*36*):

> Certainly the American Indian differs in appearance from tribe to tribe and from region to region, but underneath these local differences there is a fundamental similarity. This, too, is in favour of descent from a single, small, ancestral community. (p. 218)

Nevertheless, little by little, the physical variability of the American Indian had shown itself to be an undeniable fact, leading to differing and even contradictory descriptions and systematizations. Laughlin (*38*) presents the situation very clearly and objectively:

> Much progress has been made since the early days of anthropology in America when it was assumed that all Indians were essentially alike. The diversity of the New World populations has been well established. The significance of the diversity in terms of the evolution or development of types there or of the importation of preformed types from the Old World remains to be clarified. (p. v)

Stewart and Newman (*56*) expressed the same opinion at the conclusion of their article:

> This review of opinions regarding Indian variability has revealed the fact that the principle of Indian racial unity rests almost solely upon the outer appearance of living Indians. In so far as the Indians exhibit in common such physical characters as straight, black hair, copper-colored skin, dark brown eyes, high cheek bones, scanty beard and a relatively long trunk, they can be said to be uniform. . . . That the Indians, on the other hand, are quite variable within this racial pattern, and especially when comparisons are made in measurable dimensions, also has been shown. (p. 33)

All this would lead one to think that from 1951 on, the theory of the American homotype had been definitely discredited. While the great majority of anthropologists are of this opinion, we must not overlook the fact that some distinguished investigators differ. Ashley Montagu (*44*), for example, has written:

> The American Indians exhibit a certain basic homogeneity but at the same time are evidently characterized by an equally certain diversity of types. Owing to the lack of the necessary data it is impossible to say quite how many diverse types there may be. General impressions based on sporadically measured and photographed individuals from various groups provide an insufficient basis upon which to erect a satisfactory account of the American Indian. (p. 465)

Some years later the same author does affirm (*45*) that the Eskimos of the Arctic coast of North America form part of the group of Arctic Mongoloids while the other American Indians constitute another group composed of "an undetermined number of ethnic groups of North, Middle, Central and South America."

Coon, in his important volume on human races (*18*) devotes barely two pages to the racial characteristics of the American Indians. He says:

> The American Indians are more uniform racially than any other group of people occupying an equally vast area. In fact, they are more uniform than many peoples who occupy an area a tenth as large. All of this indicates that a relatively small number of peoples crossed the Bering Strait during the last part of the Wisconsin glaciation, and that their descendants gradually filled the uninhabited regions of the New World. They are Mongoloid in general and despite some of their peculiarities in blood groups do not necessarily merit classification as a subspecies of their own. . . . The American Indians differ from the Asiatic Mongoloids mainly in that they have less facial flatness, particularly in the nasal skeleton, and a more variable skin color. There is no valid evidence that the Indians were derived from more than one source or that they came into the New World by a route other than the Bering Strait. (p. 152)

In spite of these sporadic cases—and quite possibly there are other adherents of this theory —I believe it can be said that the beginning of the second half of the twentieth century coincides with an end to the myth of the "Indian homotype" and an explicit recognition by the great majority of anthropologists of the existence of somatic and osteological variability and heterogeneity among the aboriginal groups of

America. It is then necessary to establish the origin and the causes of this variability, bearing in mind that it in no way exceeds the limits of characterization of *Homo sapiens*.

Have we sufficient and adequate information, posterior to 1951, to be able to determine whether the differences of physical types among the Amerindians are due to prehistoric immigration from the Old World or to adaptive evolution to the new habitat? This is what we must try to analyze.

Cause or causes of biological variability of American Indians

At first glance it seems easy to distinguish two explanations for this phenomenon.

First, there is the proposal of those who accept the immigration of diverse human types, each of which represents one of the existing Amerindian "races." Stewart and Newman (*56*) describe it very clearly, attributing it primarily to those who have been concerned with American racial taxonomy:

> . . . the classifiers of Indian types operated with a strongly hereditarian bias, out of keeping with the main stream of biological thought. If explanations were given for their classifications, they were usually to the effect that each Indian "race" represented a separate migration from the Old World. . . . Implicitly, such explanations disavowed the possibility that physical changes could have occurred among New World peoples. (p. 29)

However, they themselves indicate that the various authors place different degrees of emphasis on the hereditary aspects of the prehistoric immigrants, on cross-breeding with each other, and on the environmental influence of the new habitat to explain the presence and existence of distinct Amerindian "races"; and they evaluate the greater or lesser importance that some of the most conspicuous "polyracialists"—for example, Dixon, Griffith Taylor, and Hooton—concede to each of these factors (p. 30). Even Imbelloni, one of the most devoted defenders of a complicated polyracialism, refers to only seven distinct migratory waves and on the other hand describes and localizes eleven Amerindian "races," which implies agreement that in the new habitat new racial types were formed. He even tries (*34*) to localize "the sectors and zones where the effects of mixture and hybridization occurred," although he rejects every explanation for what he called the "environmentalist creed."

Second, there is the theory of somatic variability as principally the consequence of environmental influences, which Stewart and Newman explained as follows:

> Interpretations of this sort were made largely by Americanists who had no classifications to justify, and accordingly were more willing to admit that anthropometric determinations were not always stable in changing environments. As a group, these Americanists did not deny the migrationist postulates of the classifiers, but seemed to believe that hereditarian and environmental explanations could be harmoniously blended in overall interpretations. (p. 31)

But what seems essential to me is a somewhat more careful analysis of Newman's own view (*54*), which he expresses thus:

> The adaptive responses of bodily form to environment in warm-blooded animals have led, largely in the last century, to the formulation of several ecological rules. . . . Extensive testing of these rules on human materials, however, has not been performed. For this reason, I have examined the applicability of two of the best validated of these rules—Bergmann's and Allen's—to the body forms of New World aborigines.[2] The principle behind both rules is that the maximum retention of body heat in cold climates occurs when the radiating skin surface is small relative to body mass. Since this ratio can be achieved by larger body size, Bergmann's rule holds that within a wide-ranging species, the subspecies in colder climates attain greater size than those in warmer climates. Allen's rule holds that in addition the cold climate subspecies have reduced extremities and appendages, thus further reducing the body surface. In warmer climates, following Bergmann's rule, easier dissipation of body heat goes with the low body mass body surface ratio achieved by smaller body size (p. 324)

Newman presents with the greatest objectivity a series of facts, some of which I wish to mention because his interpretation of them seems to me in certain cases to be doubtful, erroneous, and even contradictory with respect to the final comment.

1. He states (p. 312): "In mammals and birds

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[2] J. A. Allen, The influence of physical conditions in the genesis of species, *Radical Review* 1:108–140, 1877. C. Bergmann, Ueber die Verhältnisse der Wärmeökonomie der Thiere zu ihrer Grösse, *Göttinger Studien* 3:595–708, 1847.

there are a number of exceptions to these rules: 10 to 30 per cent for Bergmann's rule, calculated only from subspecies in the most contrasting climates of the species range (Rensch, 1938: 282)." Actually, the percentage of exceptions to Bergmann's rule among birds and mammals is larger than this. Rensch (*51*) mentions "for palearctic and nearctic birds I calculated 20–30% of exceptions on the average" and "for palearctic and nearctic mammals 30–40%."

2. He says (p. 313): "I am aware that in certain parts of the Old World [Bergmann's and Allen's rules] do not seem applicable. Upon superficial examination, the rules do not appear operative in Africa south of the Sahara. Yet, in Europe and the Near and Middle East and in East Asia and Malaysia, there seem to be north-south body size clines conforming to Bergmann's rule. The explanation of these discrepancies in Africa and perhaps elsewhere is not yet apparent." To admit that Bergmann's rule is not operative in Africa south of the Sahara indicates the impossibility of generalizing such a rule in support of human variability. Furthermore, its application seems doubtful in Europe, the Near and Middle East, eastern Asia, and Malaysia. How does one explain, for example, the differences of stature and body proportions in different regions of Europe, and among the Veddas and Brahmans of Bengal, the Sikhs of the Punjab, the Tapiros and Papuans?

3. In support of his thesis Newman includes nine maps showing the distribution in America of stature, sitting height (living), head size (living), cranial module (skeleton), upper face size (skeleton), morphological facial index (living), upper facial index (skeleton), and nasal index (living and skeleton); but he adds (p. 315): "The categories used in these maps are arbitrary, and in some areas the data are inadequate, but *probably* [italics mine] the maps represent reasonable approaches to the real distributions."

Then he notes that the small stature of the Eskimos constitutes an exception to Bergmann's rule but that, as "the Western Eskimo are not inferior in sitting height to the tallest Indians, their shorter stature, then, is attributable solely to their short legs. This reduction of extremity length is in accordance with Allen's rule, and *probably* [italics mine] represents an adaptation fostering body-heat retention." As to the eastern Eskimos, whose stature and sitting height are less than those of the Indians who live further to the south, he attempts to explain this by saying "*Possibly* [italics mine] the use of heavy tailored clothing in combination with factors of uncertain food supply and periodic undernutrition may cancel out the selective advantage of larger bodies in colder climates, but this cannot be demonstrated."

Continuing to note exceptions to the supposed applicability of Bergmann's rule to man in America, he mentions groups of small stature "surrounded by taller groups," such as the Yuki of northern California, the Lillouet of southern British Columbia, and the Yahgan and Alakaluf of the Magellan archipelago. With respect to the last two groups he further indicates that the sitting height has not been calculated, "but other measurements indicate they are not particularly shortlegged."

Neel and Schull (*46*) say: "In the simplest terms we may conceive of stature as being the cumulative effect of a number of genes whose actions are similar and whose effects are additive" (p. 107). "We may surmise, therefore, that under the environmental conditions in which this study was conducted, the primary cause of variation in stature is genetic" (p. 110).

Barnicot (*9*), referring to geographical variations in stature, says:

> There is a great deal of information about stature variation throughout the world, but not infrequently it is based on samples which are either very small or were selected in a way which may render them unrepresentative of the general population. . . . On the whole the pattern of stature variation throughout the world shows no very striking regularities. Both tall and short peoples are to be found in most of the major regions. (pp. 203–204)

And after specifying different cases of geographical distribution of stature, he concludes:

> This distribution of stature has been interpreted as an example of a cline with adaptive significance in relation

> to climate (Bergmann's rule). A substantial negative correlation between body-weight and mean annual temperature has been demonstrated for various regions of the world.

Harrison (*27*) also writes, in dealing with environmental interaction:

> Differences in stature are inherited, but they are also environmentally determined, since growth is profoundly affected by nutritional state, and probably by climatic factors as well. Further, the nature of the variation produced by both types of factor tend to be the same. (pp. 144–145)

We can see from this how opinions differ with regard to the problem Newman presents.

4. Farther on Newman writes (p. 323): "Of the remaining standard dimensions, *only* [italics mine] head form and relative head height show distributional patterns not readily interpreted as adaptive ones. Indeed, the earlier and marginal distribution of long heads and the apparently late arrival of low heads seems best explained by migrations of people differing in these regards. But since the diagnostic criteria of most racial classifications of New World aborigines are principally the body size and proportion traits shown here to be adaptive, it is most curious that if explanations of these classifications are attempted at all, they are in terms of a separate migration from Asia to account for each race." The "head form and relative head height" are hereditary characteristics because head breadth (dominant) and head length (recessive) enter into their determination. The same is true with respect to face height (dominant) and face breadth (recessive), broad nose (dominant) and narrow nose (recessive)—characteristics to which Newman also refers in the course of his argument.

On the other hand, the most discussed racial classification of the American aborigines (*14, 47*)—that of Imbelloni, who uses as his basis the classifications of Biasutti, Eickstedt, and Schwidetzky— takes into account cranial, facial, and nasal indices, all of them hereditary characteristics, in addition to stature. Therefore, Newman's statement does not exactly reflect the real facts.

5. Finally, Newman states (pp. 323–324): "From the foregoing, it seems clear that body build is influenced by both hereditary and direct environmental factors." And "without denying that the New World was peopled by successive migrations or infiltrations of physically differing peoples it is very likely that the American races of the classifiers are at least partly the products of adaptive changes that took place in the New World." This, generally speaking, seems acceptable; I have earlier made observations on this matter, with particular reference to the characteristics that are considered hereditary (stature, body proportions, and cranial and facial indices). The problem to be resolved should be to determine precisely—quantitatively and qualitatively—the influence that heredity and environment (in their broadest sense) have exercised on the present somatic variability of the American aborigines.

However, in his Summary (pp. 324–325) Newman generalizes about the applicability of Bergmann's and Allen's rules for the formation of the Amerindian "races" without regard to all the reservations and exceptions made in the course of his article and cited here. And in a later work (*49*) he reaffirms his opinion: "All these data should make it clear why anthropologists have paid serious attention to the application of the ecological rules to man. If anything, these rules seem to be more closely operative in man than in other species of homeotherms" (p. 104).

This is important, since other anthropologists not only accept such a generalization but tend to amplify it; Stewart (*55*), for example, says: "Thus Marshall Newman has demonstrated for the hemisphere at large that many elements of the Indian phenotype are primarily adaptive responses to environment and are distributed in accordance with Bergmann's and Allen's ecological rules" (p. 262). And, as expected, he goes on to state:

> When the first Asiatics crossed Bering Strait into America they entered a huge cul-de-sac offering every variety of environment and no forerunners to mix with. A reconstruction of what happened thereafter takes into account that the resulting population at the time of discovery *constituted a major isolate that was homogeneous, both phenotypically and genotypically.* (p. 269; italics mine)

I wish, however, to present arguments that establish categorically the true scope of this "geographical and climatic determinism." I have already said above that according to Rensch the percentage of exceptions among birds and mammals to Bergmann's and Allen's rules is very high.

In an interesting paper on the same problem, C. G. Wilber (*61*) says that "On the basis of our present knowledge the rules of Bergmann and Allen appear to be of historical or descriptive interest only and certainly are not valid generalizations for animals in the cold" (p. 332). His Summary is as follows:

> This brief and rapid survey does not postulate that climate is without effect on man. At another time the ecological effect of this variable of man will be discussed. This presentation attempted to show in a sketchy fashion the following:
>
> 1) The rules of Bergmann and Allen find little support as causal agents in modern studies of temperature regulation in homeotherms.
>
> 2) The various formal examples often cited in favor of these ecological generalizations do not support the case of the climatic determinists. One is forced to conclude that the rules just do not apply causally to animals.
>
> 3) In man the ecological forces supposed to be acting are not doing so; Eskimos were not cold, the skinny aboriginal Australians were.
>
> 4) *The rules of Bergmann and Allen have no causal role in the formation of racial differences in man. Such use of these rules on the part of some anthropologists is a source of misinformation and confusion* [italics mine].
>
> 5) Some human groups have met the demands of severe climate by technological and behavioral adjustments; the Eskimos are an example. Others have developed specific heat-conserving functional changes with no gross morphological changes; the Australian aborigines are an example.

Garn, on the other hand, concludes as follows a detailed criticism refuting Wilber (*22*):

> I know of no anthropologist so rash as to claim that temperature and the radiant heat load are exclusive or even major causes of the differences between geographical races, or that the past-century formulations of Bergmann and Allen completely solve the problems of race formation in man. I know of none who adopt the Lamarckian approach that Dr. Wilber so gleefully demolishes. But, when Wilber asserts that the "rules of Bergmann and Allen have *no* causal role in the formation of racial differences in man," I doubt very much that he intended such a sweeping and untestable counter-generalization.

Other authors confirm a divergence of opinions about the applicability and importance of Bergmann's and Allen's rules to the biological variability of the American Indian. Roberts (*53*), in an important contribution to the subject, says:

> The weight/temperature relationships here demonstrated suggest that Bergmann's rule is applicable to man. Clearer definition of "body size" is, however, necessary. Defined by reference to stature, although from the series here considered Bergmann's rule might seem to be applicable, *this suggestion is refuted by more extensive material* [italics mine]. Defined by weight, it is not only applicable but needs restatement to incorporate, with the postulated variation in size among subspecies, similar variation within the subspecies. (p. 551)

Ashley Montagu (*44, 45*) holds that such zoological rules "are to some extent also applicable to man," but adds:

> The application of Bergmann's and Allen's rules to man have been seriously and cogently questioned (Wilber), on the ground that the inadequate data has been improperly interpreted, and that in any event man has never responded to his environment in a passive manner, but has always done everything within his power to control and shape the environment to his requirements. But while this is undoubtedly true, it should be remembered that ecological rules are generalizations to which exceptions can be found in every group, but that by and large they do apply to most populations of a species. The studies of Newman and of Roberts on New and Old World human populations lend strong support to the view that ecological rules apply to man as well as to other animals.

Weiner (*58*), for his part, states that Bergmann's and Allen's rules are applicable to animal populations in general and continues: "That human body-size and shape tend to follow these rules has been demonstrated in several studies. The mean body-weight of populations in hot regions is demonstrably lower than that in temperate and cooler climates" (p. 455). But farther on in the same work, on examining genetic and nongenetic factors in climatic adjustments, he points out:

> Twin studies indicate that variations in body-shape, size, fat deposition, growth pattern, skeletal and physiological maturation are all determined by genetic constitution to a larger extent than by purely environmental factors. Certain of the population differences rest undoubtedly on distinctive genotypes or multifactorial recombinations, e.g. nose-shape, or the ratio of limb length to trunk

> length since such characters remain unaffected on change of environment. (p. 460)

Baker (*1*), also referring to the racial differences in heat tolerance, says: "These results further suggested that the differences found were not a function of transient environmental effects and may be mostly genetic in origin" (p. 303). Later (*2*) he arrives at the conclusion that "However, it is not enough to find evidence of climatic adaptation. There remains the much larger question of how climatic selection would operate on man's genetic structure to produce these adaptations" (p. 4).

In general terms I believe that Dobzhansky (*21*) best synthesizes the question.

> The environment thus instigates, foments, conditions and circumscribes evolutionary changes; but it does not decide exactly which changes, if any, will occur. (p. 408)
>
> The rules of geographic variation used to be a happy hunting ground for partisans of Lamarckism and selectionism, abounding in data interpretable as their predilections decreed. Nowadays these disputes may, I hope, be bypassed. The rules attest in any case that the environment is important as an instigator of evolutionary changes. At the same time, it must be emphasized that what has been observed are rules indeed, not laws. (p. 412)
>
> Exceptions to the rules do occur, as Rensch, who has contributed more than anyone else to their study, has duly stressed. And while these exceptions do not exactly prove the rules, they are in some ways as valuable as the rules themselves. The lesson to be derived from them is that, although the environment may guide the evolution of living things, it does not prescribe just what change must occur. (p. 413)

Waddington (*57*) offers an explanation of how this genetic-environment interaction is effected.

> We have, in fact, found evidence for the existence of a "feedback" between the conditions of the environment and the phenotypic effects of gene mutations. The "feedback" circuit is the simple one as follows: (1) environmental stresses produce developmental modifications; (2) the same stresses produce a natural selective pressure which tends to accumulate genotypes which respond to the stresses with co-ordinated adaptive modifications from the unstressed course of development; (3) genes newly arising by mutation will operate in an epigenetic system in which the production of such co-ordinated adaptive modifications has been made easy. (p. 399)

Recently the problems of human adaptability to ecological and environmental conditions have greatly interested numerous biologists and physical anthropologists. A Human Adaptability Section is part of the International Biological Program (IBP), and several international meetings have been held (Burg Wartenstein, 1964; Warsaw, Kyoto, and New Delhi, 1965) to discuss such subjects as "Human adaptability and its methodology," "Human adaptability to environmental conditions and physical stress," and a proposed regional study of high-altitude adaptation. A detailed presentation of the topics of the Human Adaptability Section appear in the ***Guide of the Human Adaptability Proposals;***[3] the results of the meetings have been published (*8, 41, 63*).

In November 1967, 60 scientists from 12 nations participated in the Conference on Man at High Altitudes, sponsored jointly by the U.S. National Committee for the IBP and the World and Pan American Health Organizations. They agreed that research on high-altitude peoples could also be applied profitably to populations living at sea level and to their medical problems, and recommended that coordinated studies be made on "problems of growth, ageing, nutrition, fertility, ***natural selection*** and epidemiology."

As for investigations completed or in progress in America on human adaptability to different conditions of heat, humidity, altitude, and latitude, a number of monographs have appeared especially on peoples of the arctic regions (Eskimos) and of high altitudes (Quechuas and Aymaras). Continuing the research program so successfully initiated by Carlos Monge, Hurtado, and other investigators at the Institute of Andean Biology in Peru in 1928, Paul T. Baker and collaborators are responsible for the most recent studies on acclimation and adaptability to different ecological environments.

Despite these advances, the problem of human variability and adaptation to different climatic conditions—that is, an evaluation of the interdependence of nature and nurture—is still unresolved. Besides Dobzhansky, whose opinion is quoted above, Neel and Schull (*46*) had already stated:

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[3] Published by the Central Office of the IBP (7, Marylebone Road, London, N.W. 1).

> It is therefore practically impossible when one is dealing with human populations to create situations which throw a sharply critical light on the relative importance of heredity and environment.

Efforts to establish principal biological subdivisions of Indians

Once the variability of the living aboriginal populations—the presence of different subspecific polytypical forms, regardless of origin and causes—is recognized and accepted, all efforts at a systematization or classification require a prior definition of the concept of race.

This is not the place or time to analyze such disputable and controversial themes as the nonexistence of human races according to Livingstone (*40*) and Brace (*17*), or the "ethnic groups" of Ashley Montagu (*44, 45*) as substitutes for racial groups, or the skepticism of Barnicot (*10*) about the possibility of defining the human "races" with the necessary precision. The classic description of the "human race" based on the typologist approach is of historical interest only. Let us stick to the modern populationist view and accept, for our purposes, any of the fundamentally similar definitions of Dobzhansky (*20*), Laughlin (*39*), Garn (*23*), Mayr (*43*), Bielicki (*13*), and others. I should like, however, to quote here some considerations presented by Laughlin (*39*), because of their clarity and conciseness.

> Race does not refer to an arbitrarily selected series of individuals, even though they may be similar in appearance, i.e., of the same type. Races may be continental or local, homogeneous or heterogeneous, large or small, ancient or recent, distinctive in appearance or nondistinctive, sharply bounded or imperceptibly bounded and possess a high or low degree of genetic relationship between members. The term race can be used at different levels of abstraction from a continental race down to a local race or even their tribal subdivisions. Local races are composed of collections of family lines which constitute breeding isolates within the larger population. No arbitrary standard of magnitude exists for the size or number of differences which must exist for a group to be termed a race. . . . For summary purposes and major contrast such high levels of abstraction are suitable. However, for research purposes it is necessary to compare the smaller, constituent subdivisions, the local races or breeding isolates of many authors. . . . Consequently, though the actual number of races in the world exists apart from observers, the number recognized depends upon the aims of the observers. (p. 90)

The concepts of geographical, local, and microraces, expounded by Garn first in 1961 and more recently in 1965, are thus comprehensible. However, before examining these possible biological subdivisions or races of the American Indian, I think it advisable to repeat what I said at the beginning—that certain contemporary anthropologists, especially Montagu and Coon, take the view that the existing variability and biological differences among the diverse groups of aborigines are not sufficient to justify a racial subdivision.

Coon (*18*), however, in referring to a work of Osman Hill on "The Soft Anatomy of a North American Indian," says: ". . . they are Mongoloids of a particular kind, just as they would be Caucasoids of a particular kind had the New World been peopled by a small band of Upper Paleolithic Europeans." Here I wish to point out that the possible presence of Caucasoid elements of European origin had been specifically noted in 1928 by Cottevieille-Giraudet.[4] Apparently certain cultural features cited by Greenman (*24*) as persisting on the eastern coast of North America tend to confirm this supposition.

The classical subdivisions of mankind from the serological point of view proposed by Ottenberg, Snyder, Wiener, and principally Boyd (*16*) mention solely an Amerindian group, to which they accord a certain homogeneity—this despite the fact that Mourant had noted evident serological differences between different populations of American aborigines.

During the last few years, the multiple investigations of Henckel, Layrisse, Lisker, Loria, Matson, Neel, Robinson, Reynafarje, Salzano, Sandoval, Sutton, Swanson, Zepeda, and others have made available greatly increased data on diverse antigens, hemoglobins, transferrins, and haptoglobins that demonstrate an obvious phenotypic and genotypic variability in these aborigines. This confirms the present conception regarding the evolutionary process of the human species and the formation of races according to populationist and dynamic criteria.

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[4] See my own *Manual of Physical Anthropology* (Springfield, Illinois, 1960, pp. 633–635) and "The Upper Palaeolithic and the New World. Comments" (*Current Anthropology*, Vol. 5, No. 4, 1964, pp. 321–322).

In this respect, I cite the latest conclusions of Matson and collaborators (*42*) on South America:

> Yet as pertaining to the present study, it seems that a sensible position of equanimity, based on the available blood group data, would permit of an hypothesis that the American Indians are not completely Mongoloid, that the present Polynesian populations are a racial mosaic and that migrants from both west and east have contributed genes to present panmictic potpourri which is Polynesia. (p. 188)

Since this variability of blood groups observed among Indian populations does not coincide with other morphological variations, one is led to think that Garn's conclusion (*23*) is correct:

> As with classifications based on morphological traits rather than on the populations themselves, artificial "serological races" add nothing to human taxonomy. The major use of blood groups in classification is in the comparison and analysis of natural populations, and in the study of natural selection in contemporary races. (p. 51)

I shall now review in some detail the racial taxonomy proposed by Garn (*23*) with reference to geographical, local, and micro-races, beginning with his definitions and continuing with his subdivision of the aboriginal populations of America.

1. The concept of geographical race, first used by Rensch, is defined by Garn as "a geographically-delimited collection of similar races," and he adds that "the existence of geographical races is due, of course, to the great geographical barriers, chief among them oceans, that formerly limited the expansion and migration of local races and protected them from introduction of different genes" (p. 14). Garn's geographical race is equivalent to the concept of continental races. On this basis he divides mankind into nine geographical races, only one of which—the Amerindian—he attributes to the New World (p. 128). This he describes with some morphologically and serologically typical traits. Earlier, however (p. 120), he alludes to the fact that some anthropologists find the morphological, serological, and biochemical differences between Mongoloids and American Indians insufficient for separating them into two geographical races and join them into a single, polytypic geographical race. I agree with him that there are slight grounds for this racial hypothesis.

2. As for local races, Garn writes: "In contrast to geographical races which are geographically delimited population collections, local races correspond more nearly to the breeding populations themselves. Whether isolated by distance, by geographical barriers or by social prohibitions, local races are totally or largely endogamous, and the very small amount of gene-flow ordinarily comes from contiguous and related local races" (p. 16).

3. What Dobzhansky defined as microgeographical races and what Lasker prefers to call breeding populations are what Garn calls microraces (p. 18); in these he makes manifest certain differences in the composition of a local race. He adds: "Micro-races, though not isolated geographically or by extensive cultural prohibitions, still differ from each other in numerous ways."

4. With regard to the value and usefulness of these concepts, Garn explains (p. 22): "Geographical races, local races and micro-races offer opportunities for very different investigations in relation to race. One is not more real or more fundamental than the other, but each provides the answer to different questions and the solution to different problems of ongoing evolution in man."

5. In referring specifically to the populations of the New World—that is, the Amerindian geographical race—Garn mentions a series of local races that "in strict contrast with geographical races are true evolutionary units. As populations such local races evolve or have evolved separately." He recognizes that "with such a diversity of local races, it is clearly impossible to make a listing of all of them" but says that "it is possible to call attention to some local

races that exemplify particular taxonomic, descriptive, or evolutionary problems" (p. 140). And before proposing a subdivision of the living aboriginal populations, he contends: "In pre-Columbian America, there were hundreds of such local races, each with its own language. We still recognize the Penobscot, the Pima, the Papago and so on. Other local races, in the Americas, as in Europe and Asia, constitute a number of isolated or semi-isolated populations as is true for the several Apache and Navaho groups now."

Only after these admonitions does Garn list five Amerindian groups of local races: North American, Central, Circum-Caribbean, South American, and Fuegian. His description of biological characteristics (pp. 144–146) is quite poor, and in the case of two of these local races he refers exclusively to cultural traits. This in spite of the fact that he qualifies them as "true evolutionary units."

Incidentally, with reference to the origin of these local races Garn takes a firm position (pp. 128–129): "Once, local differentiation in the Americas was attributed to successive waves of migrations. Today such diversity is generally accepted as the result of natural selection acting on generally small population isolates, some of whom may have a respectable antiquity of as much as 20,000 years, as shown by radio-carbon dating." [5]

I have emphasized the concepts on which Garn bases his racial taxonomy, particularly with reference to the region under discussion, because I consider his concepts proper and his principles valid. However, I disagree on the subject of the five local races he proposes, since mere observation of some of the aboriginal populations inhabiting the areas in which he locates them demonstrates their great biological (especially somatic and serologic) heterogeneity.

According to Garn's own definition, I find it difficult to understand how it is possible to include in a single local race all the aboriginal populations of South America, from the Goajiro in the north to the Araucanians in the south (latitude 10° N to 40° S), living at altitudes ranging from sea level to 4,000 meters. And this applies as well to the North American local race, ranging from the Athabaskan and Algonquian in the north to the Mayas to the south (latitude 60° N to 18° N).

Discussion

An appraisal of new techniques for interpreting the physical anthropology of the American Indian was on the agenda of the Fourth Summer Seminar in Physical Anthropology, held in New York in September 1949. I should like to start by quoting from the report of that discussion (*62*).

> Washburn suggested that much of the confusion prevalent in the American Indian field today is a result of too many varying techniques being used on the same materials, each yielding different results and hence somewhat different interpretations. He indicated that some re-evaluation of the several morphologic, metric and genetic techniques was in order if the best results were to be obtained from the available data. (p. 33)

This carefully weighed opinion expressed 19 years ago still constitutes, to my way of thinking, one of the principal reasons, though not the only one, why even now attempts at a racial taxonomy of the American aborigines yield such vague, imprecise, and even contradictory results.

I agree with Baker (*4*) that the concept of race has two uses, as a pedagogic device for teaching human variation and as a research tool for investigating biological variation, and that, as he explicitly recognizes:

> Indeed, racial classification systems are, at best, interim structures for dealing with genetic and phenotypic distances, and should be replaced by quantitative systems. It may be hoped that the comparative method will be replaced by the more accurate method of mechanism analysis. However, neither of these hopes are likely to materialize in the near future and for decades race is likely to remain a useful scientific concept. As such, it appears that the race concept will remain in human biology for many decades even though it will, undoubtedly, be a constantly changing informational construct. (p. 25)

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[5] Current data indicate that man's antiquity in America dates back 35,000 to 40,000 years. See Krieger, in *Prehistoric Man in the New World* (J. D. Jennings and E. Norbeck, eds., University of Chicago Press, 1964, p. 45).

I have expressed a similar point of view on various occasions, in discussing the usefulness and importance of anthropometry and osteometry in any attempt to determine the variability between different population groups. I reiterate this belief and in its support cite the well-documented opinion of Hunt (*34*).

> As remote racial history has ceased to be the chief excuse for field anthropometry, microevolution—especially the adaptive value of racial features in different environments—has become the core of recent studies. Schemes of measurement are being revised in terms of the 'hereditability' of somatic dimensions and factors of physical growth. Work on demography, new techniques of mapping, models of gene-flow, and racial physiology is proceeding rapidly. The unraveling of microevolution is a sufficient challenge to maintain the vitality of anthropometry for a long time to come. In particular, it offers many opportunities for the collaboration of physical and cultural anthropologists. (p. 82)

I have deliberately included in this paper many quotations—too many, the reader may think—from different workers, so as to present the most recent and contradictory opinions on the topic and thus document as objectively as possible my own view, contained in the following provisional conclusions:

1. I consider acceptable the proposal of various anthropologists for uniting all the aborigines of the New World under the denomination Amerindian Geographical Race.

2. That biological variations create perceptible differences between American Indians in diverse regions of the continent and thus necessitate a taxonomy of local races, in accord with populationist and dynamic criteria, has been fully proved and substantiated by many investigations in various fields of human biology.

3. Attempts have been made to explain this biological heterogeneity as a consequence of the diverse origin of the immigrating contingents who peopled the New World some 40,000 years ago and also as the result of a process of adaptation to different environmental and ecological conditions. At the present time, this is a point of controversy among investigators. In all likelihood the biological differentiation of the Amerindian is due to the joint action of the two. However, more data are necessary before it can be determined which is the more important.

4. The most widespread and best-known subdivisions of the American Indian in local races we owe to Garn. If, as he says, these "are true evolutionary units. As populations, such local races evolve or have evolved separately," then it is necessary to assemble a series of biological characteristics that will make it possible to distinguish the local races proposed for America.

5. The Amerindian groups of local races proposed by Garn are not precisely defined, and he appears to recognize this himself when he writes (p. 121): "Such differences in taxonomic opinion are both legitimate and salutary. They point out problem areas that need resolution. Areas of agreement on the other hand, may reflect problems long settled, or they may reflect a virtual lack of information."

What is absolutely indispensable in the immediate future is the organization on a hemisphere-wide basis of intensive, methodical, uniform biological investigations (somatic, serologic, physiological, psychosomatic, and so on) of the various aboriginal populations that permit subsequent comparative studies and finally the establishing of local races with a clear and indisputable differential biological base.

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BIOLOGICAL SUBDIVISIONS OF THE INDIAN ON THE BASIS OF GENETIC TRAITS

Miguel Layrisse

Improved techniques for the identification of new traits in the erythrocytes and in chemical components of the plasma have brought about a new era in the field of population genetics. Several of these traits have shown a distribution restricted to a single subdivision of mankind, attaining thereby the rank of gene markers; others are common to virtually all populations, although with variable frequencies; still others are fixed and/or of very low frequency. It is now possible to set up a more comprehensive "mosaic" of gene frequencies in populations, to complement the traditional division of mankind based predominantly on somatological characteristics.

Several attempts have been made at classifying mankind on the basis of genetic traits (*2, 3, 30*). Originally classifications were made by means of blood groups; later, other blood traits were also taken into consideration. In a recent classification Boyd (*3*) distinguishes 13 different human races comprising 7 major groups, one of which is the "American group" of a single race—the "American Indian." Boyd suggests that "it may eventually be possible to distinguish between North American and South American Indians serologically. . . ."

Considered as one population, American Indians in general are characterized by a high frequency of blood groups O, M, R_2, Fy^a, Di^a, and ABH secretor, and by a low frequency or absence of A_2, B, Ro, r, Lu^a, K, Le^a, and abnormal hemoglobins (*3, 21, 22*). These characteristics differentiate the American Indians so far studied from other subdivisions of mankind. However, intertribal differences suggest that Indian populations do not constitute a homogeneous gene pool as is sometimes suggested in the anthropological literature.

Characteristically, Indian populations throughout the Americas have been organized in territorial and tribal groups displaying a certain *esprit de corps* based on a distinctive cultural and linguistic homogeneity. The tribes live or have lived in a state of semi-isolation, separated from their neighbors by natural and cultural barriers that have functioned as more or less effective impediments to gene flow. Larger tribes may be divided into smaller subtribes made up of several villages. These villages are frequently quite independent of each other, with a strong tendency to local endogamy. For instance, in three recently studied villages of the Warao Indians of the Orinoco Delta, more than 90 per cent of the marriages recorded were contracted between individuals of the same local group. This high incidence of local endogamy occurred in an almost complete absence of geographical barriers to communication. Similarly high degrees of village autonomy and local endogamy were observed, although under different cultural and environmental conditions, by Salzano, Neel, and Maybury-Lewis (*28*) among the Xavante and by Salzano (*26*) among the Caingang.

Analysis of the peculiar socio-political characteristics of the Amerindian is needed so that we may learn how these subdivisions affect the frequency of genetic traits. In this connection attention must be drawn to (a) the intertribal and (b) the intratribal distribution of genetic traits.

Intertribal distribution of blood traits

One of the most striking examples of intertribal divergence in frequencies of blood traits is provided by two tribes living in the Amazon Territory of Venezuela: the Makiritare and the Yanomama. The Yanomama occupy an extensive area in southern Venezuela and northern Brazil. The Makiritare inhabit adjacent territories, mainly to the north-northeast. There are considerable differences in culture between the two tribes. The Yanomama are a Paleo-Indian society of hunters and gatherers with a rudimentary agriculture (*2, 5, 15, 14, 32*). The Makiritare have what basically can be identified as a neo-Indian culture based on slash-and-burn agriculture (*14*). Although there is evidence of interbreeding between the Makiritare and the Yanomama, the actual gene flow between them can be considered extremely limited. The comparisons of blood-group frequencies between the Makiritare and two Yanomama subtribes (Sanema and Waica) showed significant differences in practically all the blood-group systems. S, of the MNS system, is high in the Makiritare (63 per cent) but only 19 per cent in the Waica and 9 per cent in the Sanema. R_1, of the Rh system, is about twice as high in both Yanomama subtribes as in the Makiritare. The Yanomama subtribes showed the lowest frequency of R_2 ever found in Indians—6 per cent in the Waica and 3 per cent in the Sanema. The frequency of Fy^a is 83 per cent in the Makiritare and 58 per cent in the Waika and Sanema. Di^a is 16 per cent in the Makiritare, 0 per cent in the Waica, and 3 per cent in the Sanema. Since the Sanema live in close geographical proximity to the northern Makiritare, the Diego frequency can possibly be accounted for by intertribal admixture.

This is by no means an isolated occurrence. For example, differences—though less marked—in blood groups have been reported by Matson and Swanson (*17, 18*) between Maya and non-Maya groups living in Central America, Salzano (*27*) in Brazilian tribes, Matson *et al.* (*19*) in Peruvian tribes; and Matson *et al.* (*20*) in Chilean tribes. Córdova, Lisker, and Loria (*7*) did not find such divergence in Mexican Indian tribes, but their study was conducted in a large population.

Intratribal distribution of genetic traits

The study of 10 villages of the Yanomama tribe, carried out in 1966, provided excellent information on how gene frequencies may vary between tribal villages in the same area separated by relatively short distances (*2*). Five hundred and sixty-eight individuals, 72 per cent of the village populations, were examined for 7 blood-group systems, 7 serum protein traits, and 7 erythrocyte enzyme groups. The variation in gene frequencies was greater than in any other tribe examined so far under similar circumstances. Genes with low frequencies—MS, NS, R_2, Lp^a, and Ag^a—were found to have been lost in several villages, while genes with high frequencies—PGM and Se—were fixed in some villages. Genes with intermediate frequencies, such as R_1, Ms, Ns, Fy^a, and Jk^a, showed a very wide range of frequencies.

The Warao villages of the Orinoco Delta provide another illustration of gene frequency variation within the same tribe. Three villages were studied: Jobure, Sacupana, and Winikina, with populations of 190, 160, and 300 respectively (*31*). Three previous studies of these tribes had shown a very low M (less than 15 per cent) with a relatively high S (40 per cent), traveling mostly with N, which is uncommon in Indians; very high R_1; and negative or very low frequency of Di^a. The village of Sacupana was the eccentric tribe; its frequencies of the MNSs and Diego systems were completely different from those found in the other two villages and from those previously reported. The frequency of gene M was 90 per cent and of gene S only 13 per cent, and this gene was mostly traveling with gene M. It was also found that these tribes had 3 per cent of Di^a. The genealogy of the Sacupana village gave a clue to the explanation of this gene distribution. Twenty-one out of the 48 living members representing the second generation come from a polygynous family in which the father happens to be MSs and his three sororal wives Ms. All the Diego-positive cases found among the Warao of the Sacupana villages came from one family, which we were able to trace through three generations. It is interesting to observe that the members of this family also carried the transferrin D_{chl}, which is absent in all Warao individuals examined but present at various frequencies in Cariban tribes. Since the western and southeastern Warao territory is surrounded by Cariban tribes, it is likely that both Di^a and D_{chl} genes were introduced into the Warao population by Carib in early times.

The three Xavante villages studied by Gershowitz *et al.* (*9*) showed a variation of 1 to 15 per cent for the gene SN and of 24 to 39 per cent for the gene R_2; the other gene frequencies had comparable results.

Comments

Analysis of the frequency of blood groups and other blood traits has shown that the sociopolitical unit used in cultural anthropology does not always correspond to the biological unit; it appears that the population of each village rather than the whole tribe represents, in some cases, an independent gene pool. Therefore, it is not surprising that independent studies carried out in a tribe may show great divergence in gene frequencies because they are performed in different villages. Such possible variations should be kept in mind when it is necessary to decide, in trying to collect information from the literature, whether the sampling is inadequate or really representative of the population under study. According to our experience in the study of two large tribes, the gene frequency obtained by sampling the less closely related individuals from several villages of a tribe fits very well with the frequency obtained by summing up the genes counted separately in villages in which more than 70 per cent of the population was examined. But taking the test results of one local group as representative of the population at large would very likely produce a distorted picture of the total gene pool.

Blood tests in Indians are more meaningful if accompanied by other anthropological and medical studies. Demography—including a history of the tribe and formation of the villages, and a careful genealogy—provides valuable information that could serve for at least a partial understanding of the variation of the gene pool between villages. The gene pool of a village among the Warao, for instance, is the result of the fusion of two, three, or four extended families that have split off from neighboring villages. This pattern is very similar in many respects to the "fission-fusion" model observed by Neel and Salzano (*23*) among the Xavantes and by Chagnon (*5*) among the Yanomama. It is not unlikely, therefore, that variants of these patterns operate in many tribes.

It is evident that in the formation of the new village, as previously described, there are circumstances that may induce variation of the gene frequency in the following generations—the number of individuals from each family who contribute to the breeding population, sororal marriage, polygny, and others. Consequently, random genetic drift would seem a likelier explanation of the variation of gene frequencies in Indian populations than other genetic forces such as selection and gene flow. It is also probable that this dispersive process may operate similarly in the formation of hybrid populations, explaining, in some cases, gene frequencies equal to or even higher than those observed in the original populations (*14, 16*).

Even though we have considered random genetic drift as the most important genetic process operating in small Indian populations, the Indians in their habitat must be exposed to selective forces that maintain the polymorphism. So far, comprehensive studies on mortality, fertility, and diseases in Indians are scarce; however, the new orientation in the study of Indians, which includes such information, will eventually make it possible to elucidate the role of selection in the variation of gene frequencies in Indian populations.

Bearing in mind this variation, I should like to discuss finally the possibility of a subdivision of the American Indian populations that might reconcile somewhat the frequency of blood traits with other anthropological characteristics. Dr. Juan Comas has previously described here some of the attempts by prominent Americanists to classify Indians. However, none of these classifications fits the distribution of blood traits. Garn (8), for instance, included all South American Indians except the Circum-Caribbean and Fueguian in one homogeneous population. It is known that in such a large area there are populations with marked morphological, cultural, and serological differences.

Even though it is very difficult to attempt a comprehensive subdivision of the aborigines of America on the basis of genetic traits, the following taxonomic considerations can be formulated from the information available:

1. It is very likely that the Eskimo can be distinguished genetically from the other North American Indians. They are characterized by the presence of the gene B and absence of the gene Di^a. In frequency of the other blood traits they do not differ greatly from American Indians.

2. The various North American tribes are very difficult to analyze, partly because of heavy non-Indian admixture. They have been classified in one group characterized by the presence of gene A and a low frequency of Di^a; however, microdifferentiations are observed in some tribes. The Blood and Blackfeet of Alberta show a very high (60 per cent) frequency of A_1 (*6*), which distinguishes this group from the rest of North American Indians. An exploration of the possible connection between Navajo, Yaqui, and Lacandon tribes exhibiting the presence of transferrin BO-1 would be of great interest (*1*, *26*).

3. It might be possible to make a first subdivision of the Middle American Indians: the northern group including Totonacs and Mayan families, and the southern group of Chibchan or Hokan affiliations. However, the only difference found to exist between these is the frequencies of Di^a, which tend in general to be high (from 4 to 18 per cent, with a mean of 10 per cent) in the northern group, and low (from 0 to 10 per cent, with a mean of 3 per cent) in the southern group.

4. The geography of the South American continent favors the formation of microdifferentiated populations. But in spite of the variations in gene frequencies between subtribes and villages of the same tribe, it might be possible to identify genetic peculiarities that characterize the tribe as a whole with a common genetic heritage different from that of other tribes or clusters of tribes. For example, it would be interesting to investigate the relatively high frequency of A_1 among the Andean Indians, first noticed by Newman (*24*), which is not accompanied by the presence of other non-Indian genes; the possible connection, on the basis of genetic traits, between the Chilean Indians and the Polynesians, as mentioned by Matson *et al.* (*20*) in a recent study; and the possibility that the Yanomama and Warao, distinguished by the absence of the gene Di^a and by anthropological traits, could be classified, separately or together, [illegible] different from the neighboring tribes.

It is highly probable that our present hesitation in subdividing the American Indians on the basis of genetic traits will be overcome in the near future with the increase of gene markers and the use of mathematical models in which all the variables found in a tribe can be computed.

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DISCUSSION

Moderator: Dr. Comas' paper emphasized the variability existing in the American Indian on the basis of morphological characteristics. Dr. Layrisse has now emphasized the variability on the basis of genetic traits, but has also directed our attention towards an aspect of the Indian that is increasingly occupying the thinking of those of us who work with the Indian: that within a relatively undisturbed tribe, there is a great deal of genetic variability from one part of the tribal distribution to the next. The geneticist has tended in the past often to think of a tribe as a kind of homogeneous beanbag, but it now becomes clear that this is not the case and that there may be very significant differences between, say, one part of the Yanomama territory and another.

The discussion of these two papers will be opened by Dr. Arends.

Tulio Arends

The physical variability of the American Indian was recognized even by the conquistadors. In 1530, Federman described a tribe of dwarfs in western Venezuela "not taller than five to six handbreadth" and found the Guayqueri in the eastern part of the country to be "people as black as coal."[1] Anthropometric studies have accumulated a great many data on stature, cranial and cephalic indexes, body proportions, and so forth, which multiply these differences. If to the somatic characteristics are added the many inherited biochemical and hematological variations known today the diversity of the American Indian becomes infinite. As Dr. Comas pointed out, to determine which of these characteristics stems from nurture and which from nature makes the problem more complex.

However, if we accept that the original population of America derived from a small group of hunters-and-gatherers from eastern Siberia who crossed the Bering Strait during the latter part of the Wisconsin glaciation and whose descendants gradually became masters of an uninhabited continent, we can understand better the great influence of environmental factors upon the American Indian. There is still the question of contact with other racial groups during the pre-Columbian era, especially with Caucasoids of European origin as mentioned by Dr. Comas.

The great progress made lately in the study of blood groups, serum protein groups, and enzyme polymorphism nullifies the idea expressed some years ago: "Type one Indian and you have typed them all; so much are they alike in their blood groups."[2] The view that serology has not added anything new to human taxonomy[3] is also no longer tenable. On the contrary, we who work in this field believe that the amount of information already accumulated will play an important role in the reinterpretation of the American Indian taxonomy and is laying the foundations for a future molecular anthropology. Boyd has said: "The genetic method has, in short, made contributions to anthropology that morphologic methods could hardly have made, and is providing a new and fundamental basis for the study of race."[4]

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Important factors in the study of the American populations

To establish biological subdivisions of the American Indians based on the study of their genetic characteristics, the best-known methods of population genetics should be used, with special emphasis on the following points:

1. It is advisable to study as many genetic traits as possible, in order to avoid biased interpretations and rule out gene drift, especially when dealing with small populations subjected to epidemics, wars, displacements, and other events that might have modified the frequency of some traits.
2. Indian villages should be studied intensively from a historical and genealogical point of view, for the purpose of evaluating the so-called "founder effect" or pursuing a foreign gene introduced through a nonapparent admixture.
3. The "fission-fusion" phenomenon, which according to Neel *et al.*,[5, 6] is not infrequent in the dynamics of Indian populations, could explain some odd findings in the prevalence of genetic traits.

Common characteristics in the American Indian

From a genetic standpoint, most of the American Indians share certain characteristics that differentiate them from other racial groups (Table 1), as Dr. Layrisse has already mentioned. We may say that the authentic South American Indians are all of group O; usually lack r and R^0 gene complexes of the Rh system; are negative for the main factor of Kell, Lewis, and Lutheran blood group systems; have only Hb-A; and are not deficient in glucose-6-phosphate dehydrogenase. [7, 8] It could be added that they are equally negative for, or have a very low frequency of, the atypical pseudocholinesterase gene E_1^a.[9, 10]

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TABLE 1. Characteristics common to American Indians

GENETIC SYSTEM	CHARACTERISTICS COMMON TO ALL SUBJECTS	EXCEPTIONS
ABO	Group O	Navajo, Pueblo, Blood, Shoshone, Blackfoot (U.S.A.)
Rh	r and R^0 absent	Apache, Pawnee (U.S.A.); Tarasco, Chontal (Mexico); Itza Maya (British Honduras)
Kell	kk	
Lewis	Le(a—)	
Lutheran	Lu(a—)	Diegueño (U.S.A.); Brazilian (Brazil)
Hemoglobin	Hb-A	Nahua, Chiapas (Mexico); Oyana, Arawak (French Guiana)
G–6–PD	Absence of deficients	Oyana, Carib (Dutch Guiana)

Where published reports give different characteristics from those in Table 1, the reason is mainly the small size of the sample studied (in some cases fewer than 50 individuals) or admixture with other racial groups. Nevertheless, there are some tribes for which the results also disagree although they are unmixed and a large enough sample was examined. These populations deserve to be thoroughly studied for a good explanation of this exception, and this could be the so-called heterogeneity of American Indians. (The same may be said of the figures in Table 2.)

Quantitative differences in the genetic systems of the American Indian

In certain genetic characteristics the American Indian is distinguished only by a quantitative frequency widely different from that of other racial groups (Table 2). The interpretation here is really difficult because different samples from the same population have sometimes given different results. For example, when the haptoglobin system of two Makiritare samples was studied,[11] results for Hp^1 as dissimilar as 0.610 and 0.342 were found. These results depend greatly upon the condition of the sample obtained. In addition to the exceptions mentioned by Dr. Layrisse, there are tribes that have given frequencies different from those mentioned even though the sample was larger than 100. Again, here are many populations that could be very usefully studied with the available genetic, biochemical, and hematological battery of tests.

Differential characteristics of some Indian tribes

Finally, there are some genetic markers that identify certain tribes (Table 3). The antigen Di(a+) of the Diego system, for instance, is a Mongoloid characteristic. The same may be said of the transferrin D_{chi}. The absence of those markers in many tribes could be an indication of gene drift, fission-fusion, or founder effect. Furthermore, the transferrin system has another variant ($Tf\text{-}B_{0-1}$) that distinguishes the North and Central American from the South American Indians.[12]

Human alloalbuminemia, or the presence of albumins with electrophoretic migration different from the normal, expressed by either one

TABLE 2. Quantitative differences in some genetic systems of American Indians

GENETIC SYSTEM	CHARACTERISTIC	EXCEPTIONS
MNSs	Gene M $\leqq 0.7$	Jicaque (Honduras)
Rh	Gene complex $R^2 > 0.35$	Seneca, Pima (U.S.A.); Jicaque (Honduras); Miskito, Sumo (Nicaragua); Maya, Mazatel (Mexico); Ticuna (Peru); Quechua (Ecuador); Xavante (Simões Lopes, Brazil); Waica (Venezuela)
Duffy	Gene $Fy^a \leqq 0.6$	Guaymi (Panama); Jicaque (Honduras); Mazatel (Mexico); Xavante (Brazil)
ABH Secretor	Gene Se $\leqq 0.8$	Xavante (Brazil)
Haptoglobin	Gene $Hp^1 \leqq 0.5$	Yupa (Venezuela); North Athabascan, Tlingit, Navajo (U.S.A.)
Gammaglobulin	Gene $Gm^a = 1.0$	Xavante (Brazil)
Inv	Phenotype Inv(a+) $\leqq 0.6$	
Gc	Gene $Gc^1 > 0.65$	Xavante (Brazil)

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[11] Arends, T. Los grupos séricos humanos determinables por métodos electroforéticos: su distribución en Sur América. *Acta Cient. Venez.* Suppl. 3:162–178, 1967.

[12] Arends, T., G. Brewer, N. Chagnon, M. L. Gallango, H. Geroshowitz, M. Layrisse, J. Neel, D. Shreffler, R. Tashian, and L. Wietkamp. Intratribal genetic differentiation among the Yanomama Indians of Southern Venezuela. *Proc. Nat. Acad. Sci.* 57:1252–1259, 1967.

TABLE 3. Genetic markers in some Indian tribes

GENETIC SYSTEM	CHARACTERISTIC	TRIBES IN WHICH IT HAS BEEN FOUND
Diego	Di(a+)	North American, Central American, South American
Transferrin	Tf B_{o-1}	Navajo (U.S.A.); Yaqui, Cora, Chinantec, Lacandon, Tzotzil, Zapotec (Mexico) Quiche (Guatemala)
	Tf D_{Chl}	Irapa, Macoita, Paraujano, Bariri, Shaparu, Motilon, Piaroa, Warao, Guahibo, Guayqueri (Venezuela); Paez (Colombia); Quechua (Peru)
Albumin	Naskapi	Naskapi, Montagnais (Canada); Sioux, Athabascan (U.S.A.)
	Mexico	Zapotec (Mexico)
	Maku	Maku (Brazil)
	Warao	Warao (Venezuela)

(homozygous form) or two components (heterozygous form), is a system that could be very useful in the American Indian taxonomy. This system, which at present is the subject of intensive study, has made it evident that certain tribes in Canada and the United States have an albumin variant faster than normal (albumin Naskapi). In Mexico a tribe was found to have an albumin slower than normal (albumin Mexico). In Brazil a faster variant (albumin Maku) and in Venezuela a slow variant (albumin Warao) different from those previously mentioned were found.

Summary

1. Some characteristics are shared by all the Indians of the American hemisphere. Populations that seem to be an exception should be investigated by the best serologic, immunologic, electrophoretic, and chromatographic methods at hand in order that characteristics reported in the past, when modern methods and reagents were not available, may be rechecked.

2. The differentiation of populations by frequency gradients of genetic traits should be the subject of careful studies, about their formation, cultural level, and kinship organization. Since these populations have been under physical, pathologic, and war pressures, it will always be very difficult to evaluate how much these agents have influenced the distribution of the inherited variations.

3. Some genetic markers—for example, Di (a+) group of the Diego system, transferrin D_{Chl} and B_{0-1}, and albumin Naskapi, Mexico, Maku, and Warao—have been found in only some Indian tribes. The characterization of each of them and their differentiation from other traits of the same system should be determined by the best biochemical and physico-chemical methods available.

4. The finding of some genetic traits (transferrin D_{Chl}, Diego group Di(a+)) among tribes is positive proof of present or remote relationship. Their absence in other tribes could be explained by gene drift, founder effect, or fission-fusion.

5. The more genetic systems are discovered, the more urgent it is for them to be studied in American Indians before these populations disappear through cultural and economic pressures or through epidemic, endemic, or sporadic pathological processes.

Rubén Lisker

The data presented today by Dr. Layrisse clearly show that present-day Amerindian populations do not have a homogeneous genetic constitution. However, the task of subdividing them on the basis of genetic traits is not an easy one, especially if to taxonomy is added an interest in interpreting or understanding the reasons for the differences encountered. A general cause of this difficulty is that the Amerindians as a group have characteristic gene frequencies for many traits that do not allow further subclassification. A good example would be blood group O and the Rh-negative phenotype. A reasonably "pure" Indian population should have close to 100 per cent of group O individuals and no Rh-negative subjects; deviation from this pattern would be generally interpreted as the result of non-Indian admixture.

Even so, some genetic traits do show enough variability in apparently unmixed Indian groups that attempts at taxonomy are possible. The difficulty here—and I should like to stress this point—is what the classification means. I feel sure that this consideration explains, at least in part, Dr. Layrisse's understandable hesitation in subdividing Amerindians.

I want to consider briefly some examples of the difficulties I have mentioned. The Diego antigen appeared to be an excellent marker of Indian groups, especially suitable for taxonomical purposes since it is not present in other ethnic groups (except some Mongoloids) and shows enough intertribal variability to allow for subdivisions.[1] However, it soon became apparent that the correlation of its distribution with either geographical or cultural criteria was not too good,[2] and furthermore, although its absence in Negroes and Caucasians is accepted, some hybrid populations in which they certainly contribute to the gene pool have higher Diego frequencies than the original Indian populations.[3, 4, 5] This is clearly seen in Table 1; seven out of the nine hybrid groups studied on the coasts of Mexico have higher Diego frequencies than most of the pure Indian tribes. Before learning the meaning of this phenomenon, one would hesitate to use this marker to subdivide Amerindians. Incidentally, I would say that although these populations show less variability than those Dr. Layrisse has studied, there is still no pattern to Diego frequency because individuals with the same linguistic affiliation show very marked differences in frequency.

TABLE 1. Frequency of Diego antigen in 18 Indian tribes and 9 coastal mestizo groups of Mexico

GROUP	PHENOTYPE RANGE (%)	FREQUENCY (%)
Indians [a]		
5 tribes	2–8	
9 tribes	12–19	
3 tribes	20–24	
1 tribe	40	
Mestizos [b]		
West Coast		
Ometepec		28.2
Cuajinicuilapa		26.3
San Pedro M.		23.4
Pochutla		31.3
East Coast		
Saladero		4.1
Veracruz		32.8
Paraiso		32.1
El Carmen		38.6
Tamiahua		3.5

[a] More than 50 individuals studied in all but two tribes.

[b] More than 90 individuals studied in each location.

An additional difficulty stems from the fact that in the study of Indian tribes with some degree of non-Indian admixture, the variation in the gene frequencies of some of the traits does not follow the expected trend. The following examples will clarify this statement. In the investigation of Mexican Indian groups, four had evidence of Negro admixture, as judged by the presence in them of a few individuals with the sickle-cell trait and/or red cell glucose-6-phosphate dehydrogenase (G-6-PD) deficiency.[6] These groups would be expected to have higher frequencies of the M gene and the cDe chromosome and lower of the Fy^a gene than the other tribes. As can be seen in Table 2, neither of these expectations was confirmed.[5] It would be also logical to expect the Hp^1 gene frequencies of the Indian tribes with evidence of Negro admixture to be higher than in the rest. Table 2 shows that this is not the case, and that in fact the Cora have the lowest Hp^1 gene frequency in our material.[7]

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[1] Layrisse, M., and J. Wilbert. El antígeno del sistema sanguíneo Diego. Caracas, Sucre.

[2] Comas, J. Significado de la presencia del antígeno Diego entre los Amerindios. *Anal. Antropol.* (Mexico) 2:89, 1965.

[3] Lisker, R., A. Loría and M. S. Cordova. Studies on several genetic hematological traits of the Mexican population. VIII. Hemoglobin S, glucose-6-phosphate dehydrogenase deficiency, and other characteristics in a malarial region. *Am. J. Hum. Genet.* 17:179, 1965.

[4] Rodríguez, H., E. Rodríguez, A. Loría, and R. Lisker: Studies on several genetic hematological traits of the Mexican population. V. Distribution of blood group antigens in Nahuas, Yaquis, Tarahumaras, Tarascos and Mixtecos. *Hum. Biol.* 35:359, 1963.

[5] Cordova, M. S., R. Lisker and A. Loría: Studies on several genetic hematological traits of the Mexican population. XII. Distribution of blood group antigens in twelve Indian tribes. *Am. J. Phys. Anthropol.* 26:55, 1967.

The recently described polymorphism of the albumin locus seemed to have taxonomical value, since in all the initial reports it followed an orderly geographical distribution.[8] Albumin variants are rare in all groups so far studied except the Amerindians, and it appeared that a fast variant known as albumin Naskapi[9] was prevalent only in Eskimos, Canadian Indians, and northern North American Indians, while the slow variant known as albumin Mexico[10] was present in southern North American Indians and in some Mexican Indian groups and mestizos. The only information from South America relates to two groups of Peruvian Indians, another from Brazil, and a fourth from Brazil and Venezuela (the Yanomama), where no variants were found except in the last group.[11] This pattern has already at least three exceptions: the Huasteco in northeast Mexico and the Chol in southeast Mexico have a fast variant that has electrophoretic mobility similar to the Naskapi type and may be the same, and the variant recently encountered in the Yanomama is fast but different from the Naskapi.

It is also interesting to remark that whereas the presence of albumin variants in Eskimos might lead to an attempt to group them together with other Indian groups, it is evident, as stated by Dr. Layrisse, that the only American aboriginal population that clearly deviates from the rest of the Amerindians in the distribution of several genetic traits is the Eskimos.

It seems to me that, regardless of the rather erratic distribution of some traits in the Amerindians, and despite the fact that the precise reason for this is unknown, it is important to continue the characterization of the genetic constitution of the Amerindians, quite apart from the question whether or not it can be used at this point for meaningful subclassification. I

TABLE 2. Comparative frequencies of genes M, Fy^a, Hp^1, and chromosome cDe+cde in groups having some cases with Hb.S and /or G-6-PD deficiency with other tribes investigated for these markers

		NO. CASES		GENES		CHROMOSOME	Hp^1
GROUP	NO.	Hb.S	G–6–PD	M	Fy^a	cDe + cde	GENE
Huasteco	235	0	3	—	—	—	0.51
Nahua	141	0	4	0.77	0.64	0.04	0.44
Chontal	101	2	2	0.69	0.67	0.11	0.57
Cora	96	1	0	0.76	0.99	0.06	0.40
Others (range)		0	0	0.65 – 0.79	0.50 – 1.00	0.03 – 0.12	0.44 – 0.65

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[6] Lisker, R., G. Zárate, and A. Loría. Studies on several genetic hematological traits of Mexicans. IX. Abnormal hemoglobins and erythrocytic glucose-6-phosphate dehydrogenase deficiency in several Indian tribes. *Blood* 27:824, 1966.

[7] Lisker, R., G. Zárate, and E. Rodríguez. Studies on several genetic hematological traits of the Mexican population. XIV. Serum polymorphisms in several Indian tribes. *Am. J. Phys. Anthropol.* 27:27, 1967.

[8] Melartin, L. Albumin polymorphism in man. Studies on albumin variants in North American native populations. *Acta Path. Microbiol. Scand.* Suppl. 191, 1967.

[9] Melartin, L., and B. Blumberg. Albumin Naskapi, a new variant of serum albumin. *Science* 153:1664, 1966.

[10] Melartin, L., B. Blumberg, and R. Lisker. Albumin Mexico: a new variant of serum albumin. *Nature* 115: 288, 1967.

[11] Weitkamp, L., J. Chagnon, J. Saave, F. Salzano, and J. Gall. Serum albumin variants in American and New Guinea indigenes. *Clin. Res.* 16:298, 1968.

offer at least two important reasons: one, that this will provide the geneticist of tomorrow with a good baseline for understanding population dynamics in the future; the other, that longitudinal study of the correlation between the genotype and the biological development of a population, for which at least some Indian groups are quite adequate, might significantly contribute to our understanding of the reasons for the variability observed today.

General Discussion

Bier: I would like to know whether the albumin variants are antigenically identical.

Lisker: When I say there are two identical albumins —I am only saying that they have the same electrophoretic mobility. Nobody has done an amino acid sequence, so that is all we know. Whether or not it is the same mutation is unknown.

Bier: Before doing the sequence, wouldn't it be easier to make an Ouchterlony plate to see whether or not they have common determinants?

Lisker: It has been done, and no differences have been found.

Salzano: I think that the studies on Venezuelan Indians in different stages of cultural evolution, and also the researches of Dr. Lisker in Mexico, are very important since a pattern appeared among the Brazilian groups that can be tested. Three stages can be visualized. The first would be that of the hunting-gathering tribes, who have a kind of fission-fusion model. That is, the pattern is one of exchange between groups carried out mainly by more or less large subgroups of the tribes. In what I would call the second stage, the groups would rely more on agriculture but would not yet have a critical size. These would conform more to what Sewall Wright calls the island model; that is, their populations would be small and separated by large distances. *A priori,* more variation would perhaps be expected in the second groups than in the first. The third stage would be that reached by tribal groups in Mexico. The genetic structure would also be different then; it would be more likely to be the one that Sewall Wright calls the isolation-by-distance model. This means that the genetic differentiation of these groups would follow a different pattern from the other two, with less variability. In a general way, I believe the genetic and demographic data are substantiating this. But, of course, in order to be sure we need more data on different groups, in different ecological situations.

I do not think that genetic data *per se* would be very important for the differentiation of Amerindians unless they were correlated with this kind of data. For instance, the selection acting on groups in the second stage is probably quite different from that acting on the other two. This was clearly indicated by Dr. Dubos in his book *Man Adapting.* The nutrition of these groups would become somewhat more limited; they would rely on just a few crops that can provide enough calories but make for an unbalanced diet. I could give many other such examples to show that the situation between groups in these three stages is quite different and that if data from populations at these different cultural stages are mixed, a meaningful picture is probably impossible.

Waterlow: Dr. Neel, is it really proved that all these things—albumin variants, haptoglobin variants, red-cell variants—are genetically determined or is that an assumption?

Moderator: I believe the speakers could take care of this point better than I could, but very briefly the answer is yes, it is proved. However, I should qualify this a bit. The genetic basis cannot be determined from a simple population survey. Family studies are required. And in every instance in which a variant has been described today, there have been studies that show it is transmitted according to Mendelian laws within family lines. And this kind of evidence is necessary before we can speak of a variant with any assurance.

THE AMERICAN INDIAN IN THE INTERNATIONAL BIOLOGICAL PROGRAM

James V. Neel

Administrative background

As is a matter of common knowledge, in 1960 the International Council of Scientific Unions (ICSU) took the initiative in the promulgation of an International Biological Program. As originally envisioned by ICSU, the objectives of this program were the world-wide study of (1) organic production on the land, in fresh waters, and in the seas, and the potentialities and uses of new as well as existing natural resources; and (2) human adaptability to changing conditions. A Planning Committee appointed by ICSU in turn established a number of subcommittees to consider various aspects of the program and to elaborate a more extended protocol. These various subcommittees combined their efforts into a series of suggestions and recommendations, which the Planning Committee coordinated and submitted to ICSU in November 1963. ICSU, after approving these suggestions and recommendations, then called upon the various countries having representation in it through national societies to formulate national programs. The timetable for IBP, several times revised, consists of a "planning phase," which officially terminated in mid-1967, and an "operational phase," to continue until mid-1972.

It has been clearly understood from the outset that the various national committees were in no way bound by this international formulation—it was designed merely to provide convenient guidelines. Thus far ten countries in North, Central, and South America have established committees to formulate national programs, and four other countries may do so soon. It will be the purpose of this presentation to attempt to point out the manner in which a variety of types of studies of the American Indian fall within the framework of the IBP and especially the section concerned with human adaptability. In fact, the Indian provides the Americas with a unique resource in IBP-oriented studies and at the same time presents interdigitating medical and humane challenges that are worthy of the consideration of this Organization.

At all the various levels of consideration of human adaptability, it has been clear there are two interrelated components: the genetic, which sets the limit of the organism's ability to respond, and the physiological, which determines the range of response available to the organism, given its genetic set. At one extreme in the spectrum of interests that can be brought to bear on human adaptability is environmental physiology, and at the other is population genetics—and in between there is room for many aspects of modern medicine and anthropology.

From the earliest considerations of most of the various committees and subcommittees concerned with human adaptability, it has been apparent that the surviving primitive populations afford an opportunity to study physiological and genetic adaptations potentially quite different from those of highly civilized groups. Parenthetically, although our cultural orientation is such that we tend to view many of these surviving primitive groups as representing extremes of adaptability, the fact is that they have lived under their conditions far longer than we under ours. Viewed from their vantage point, we are the ones pressing for new limits of adaptability, and it is only an article of faith that we will adapt to our man-made environment as successfully as they have done to theirs. We must note in passing that some of the adaptations of these groups are complex and ingenious, and that the term *primitive* as commonly used is something of a misnomer, amounting to "preliterate with a relatively simple technology."

In late 1962 the World Health Organization convened in Geneva a Scientific Group on Research in Population Genetics of Primitive Groups. Its purpose was to consider studies of populations of unusual genetic interest, with particular reference to the surviving hunting and food-gathering groups of the world, to advise on the most suitable populations for such studies, and to draw up appropriate research designs. The pertinence of this to IBP-related developments is obvious. In the preliminary inventory of these societies published in the report of the Scientific Group, various South American tribes figure prominently (*26*). This report, incidentally, was updated in the summer of 1967 and a revised version has just been issued as WHO Technical Report 387.

A recurrent consideration in the consultations of both IBP-related and WHO-related groups has been the rate at which these primitive groups are disappearing. It can be said without exaggeration that ours is probably the last generation privileged to study such groups in a relatively pristine state. If there are important scientific lessons to be learned—and the WHO document would seem to develop that case adequately—then there is an urgency to this problem not shared by many other areas of research.

The dimensions of Indian-oriented studies

These are days in which those of us who are concerned with human biology view our species as beset by a growing number of pressing problems of our own making: the population increase, trace chemicals in foodstuffs, air pollution, water pollution, the so-called stress diseases, cultural alienation, to mention only a few. Each of these problems has reached such a magnitude as to call for concerted action on a hitherto unknown scale. But I submit that behind these immediate problems lurks a far larger issue. Now that man so largely controls both the environment and his reproduction, is there any long-range objective more important than that we understand the genetic potentialities of the individual and the species, the manner in which this potential interacts with an environment that is increasingly man-made, and the rate of genetic change in response to a changing environment? You will recognize that I have only restated in a different context the need to understand the genetic aspects of human adaptability. The American Indian offers prime research material for this objective. The principal lines of attack can be conveniently summarized under three headings (see *12*).

1. *Population structure and natural selection in primitive man.* One of the basic tenets of biology is of course a belief in the role of natural selection in shaping each species to its ecological niche. As biologists we believe that natural selection has guided human evolution and will continue to guide the evolution necessary for a successful genetic adjustment to a changing world. It is clear that modern man evolved under circumstances approximating the conditions found in primitive cultures. Otherwise stated, there is no evidence that the developments of the past few thousand years have yet resulted in any very significant modifications of man's genetic potential. It follows that one way of understanding the genetic impact of our changing environment is from the baseline supplied by detailed knowledge of primitive man in his primitive ecosystem. The simple fact is that at present we know virtually nothing about how selection works in man. What few insights we have are largely based on the results of artificial selection with experimental animals and deal with traits, such as extremes of egg production or milk yield, considerably more important to us than to the animal species involved.

What the geneticist terms "population structure" places limits on the effectiveness of natural or artificial selection. Thus, hand in hand with efforts to understand selective pressures must go efforts to understand the genetic structure of populations. To the geneticist, this means patterns of mating, fertility, and survival; amount of inbreeding; migration patterns; mutation rates; and so on. Although some data on these subjects are to be found in the voluminous anthropological literature, in the main what is available simply lacks the precision and depth necessary to the precise statistical treatments of the geneticist.

Even with all the facilities of modern science and the conveniences of our culture, such comprehensive studies remain extremely difficult in civilized countries. How much more difficult are they, then, in the remote primitive populations—problems of logistics and communication being what they are. Even so, from our own experience of the past several years and that of several other groups with kindred interests, it is clear that a variety of types of data relevant to population structure can be collected.

In1962, aided by a grant from this Organization, Dr. Francisco Salzano of the University of Rio Grande do Sul, Brazil, and I, with several collaborators, undertook a pilot study on the Xavante of the Brazilian Mato Grosso (*13*). These and further studies on the Xavante, in 1963 and 1964, raised a number of rather basic questions for population genetics (*14*), but it was felt that the Xavante were neither a large enough group nor sufficiently undisturbed for a critical approach to these questions. Accordingly, after a careful consideration of the possibilities, a second study was undertaken, concerned with the Yanomama Indians of Venezuela and Brazil and involving as principal colleagues—in addition to Dr. Salzano—Drs. Miguel Layrisse and Tulio Arends of the Venezuelan Institute for Scientific Research (IVIC) and Drs. William J. Schull and Napoleon Chagnon of the University of Michigan. We have now been in the field on three different occasions. The composition of the team varies from year to year, but ideally it includes an ethnologist, a linguist, a physical anthropologist, a geneticist, and several physicians. Although the precise nature of the data collected depends upon local circumstances, under optimal conditions we assemble as complete demographic and genealogical data as possible; conduct physical, dental, and anthropological examinations; and obtain blood, urine, saliva, and stool specimens for a variety of genetic and other determinations. The effort to understand better man's interaction with his environment carries far beyond the identification of genetic traits—we are fortunate to have the collaboration of such groups as the Communicable Disease Center of the U.S. Public Health Service, the Virus Institute at IVIC, and many specialized laboratories at the University of Michigan.

This is not the time for a detailed review of the findings on either the Xavante or the Yanomama, which in any event must be regarded as preliminary. Not at all surprisingly, both the population structure of these people and the biological pressures on them are qualitatively and quantitatively different from those of civilized groups. These are populations in which infertility is rare, practically all women contributing to the next generation; in which effective fertility seems intermediate, and infant and childhood mortality also intermediate; and in which the variance of male reproductive indices is significantly greater than of female (*15*). A surprising amount of genetic microdifferentiation between villages has been encountered (*1*), originating, we believe, in the fission-fusion pattern that dominates village proliferation and results when a new village comes into being in a very non-random sampling of the tribal gene pool. We reason from this that if new tribes originate in general as new villages do, then chance may have played a greater role in much measurable human genetic variation than heretofore seemed likely. The roles of traumatic, infectious, and nutritional disease in determining survival are far different than in the next step on the cultural ladder—that is, in relatively densely populated agricultural societies.

We do not pretend to understand as yet the full genetic implications of all that we see. One reason for our lack of understanding is that a substantial portion of the mathematical theory of population genetics is currently being reworked. A second reason is that the data are so complex that precise mathematical formulations do not appear imminent, and we are being forced to involved computer simulation programs to explore the genetic consequences of the differences between these societies and our own. However, even at this stage it is very clear that it will be possible to define objectives and test hypotheses in a way that will justify the effort expended.

I mentioned above that a substantial portion of the mathematical theory of population genetics is being reworked. One of the most provocative developments in the biomedicine of the past twenty years has been the realization of the extent of man's concealed genetic variability, as revealed by modern biochemical techniques. Many of the newly recognized variant types occur in populations in such frequencies that we speak of the underlying systems as genetic polymorphisms. The maintenance of such polymorphisms is thought to require selective pressures of one kind or another; not only is the nature of these forces unknown, however, but under the previous formulations the mortality and fertility structure of our population scarcely seems adequate to accommodate the necessary selection, and new formulations are being considered (*7, 9, 25*). Understanding how all this variability is maintained is one of the great challenges to the human biologist (see discussions in *8* and *16*). Now, the genetic polymorphisms are well represented in all the primitive populations studied to date. This is where they came into being. Accordingly, it seems self-evident that, despite the inherent difficulties, we must make every effort to conduct studies in depth of population structure and the relationship at this cultural level between the polymorphisms and specific biological attributes.

There is an urgent scientific need for similar studies, while the opportunity exists, on as many as possible of the other unacculturated groups of Indians. None of the surviving primitive Indian communities can safely be labeled "typical"—each may be unusual in one or several respects. Only from the perspective of numerous studies in depth can we begin with any assurance to factor out the significant common denominators that have guided human evolution. Already parallel studies are in progress on such groups as the Cayapó of Brazil by Salzano and colleagues and the Kashinawa of Peru and Brazil by Johnston and colleagues, as well as in other parts of the world, and more are to be hoped for.

2. *The rate of human evolution, i.e., genetic diversification.* Despite the many unanswered questions discussed earlier in this session by Professors Griffin, Cruxent, and Laughlin, the date and place of arrival of the Indian in the Americas is more accurately known than the corresponding information for any other major subdivision of mankind. In view of what can be deduced concerning population density in northern Europe 20,000 years ago, we may surmise that the total number of immigrants across the Bering Bridge, whether they came in one or in several discrete waves, was not large. The genetic characteristics of this original group will of course be forever conjectural, but let us assume that the tribal population(s) concerned had the range of variability we see in such a group as the Yanomama. (I am assuming in this discussion that the contribution to the gene pool of individuals reaching the Americas other than by the Bering Bridge is negligible.) Once here, extension throughout the Americas seems to have been rapid. In this extension, the diversity of habitats explored and occupied represents most of the extremes of the habitable portions of the earth. If we make the assumption of a reasonably homogeneous beginning, then the diversity of types of Indians at the time of the rediscovery of this continent five centuries ago constitutes an unusual opportunity to measure the rate of human evolution.

Much of the early thinking on this topic drew on morphological characteristics, and these, as Professor Comas has just demonstrated, still remain valid and pertinent. However, in recent years the newly discovered polymorphisms mentioned earlier have provided a powerful battery of objective indicators of genetic distance. Dr. Layrisse has just provided us with an overview of how great the range is with regard to a variety of specific systems. In theory, for any given genetic system the position of each Indian population with respect to all the others can be defined by a point in a space that has one less dimension than there are alleles in the system, the precise position in this space being determined by the frequency of each allele. Further in theory, when many different genetic systems are considered simultaneously, the position of each population vis-à-vis the others can be plotted in a space that now has as many dimensions as there are genetic systems. Previously, the computations when a considerable number of systems was involved were as laborious as this description sounds (*6, 22*). But the advent of the modern computers has dramatically altered the situation, and now a number of groups are developing computer-based measurements of population distance (see *3*).

Although considerable progress has been made, there is still a great deal to be done in the way of characterizing the genetic attributes of the relatively pure Indian groups, as a basis for exploring the evolutionary problem discussed above. Incidentally, the HA/IBP/IUBS emphasized the need for continuing efforts at the genetic characterization of all the human strains, so that this type of investigation of the Indian fits readily into a larger framework. The published data on the Indian are widely scattered, no survey having been attempted since those of Mourant (*10, 11*) and Salzano (*18*). Dr. Richard Post, Dr. William Schull, and I have now completed a compilation of all publications prior to November 1967 that deal with the principal genetic polymorphisms known to occur in the Indian.[1] The rate of discovery of new polymorphisms being what it is, any such compilation is soon dated, but for the specialist it at least provides a nidus for updating. The 271 papers on which the tabulation is based attest to the strong interest in this subject. Since the data on unmixed Indian groups have in most contexts far greater interest than the data on mixed, an effort has been made to recognize three levels of miscegenation: (1) essentially none; (2) known, but probably accounting for less than 5 per cent of the gene pool; and (3) greater than 5 per cent. The tabulation contains data on the following polymorphic systems (these being the ones on which there seemed enough data to warrant a tabulation): ABO, Rh, MN, Kidd, Duffy, Diego, P, Lewis, haptoglobins, transferrins, and Gm. Gene frequencies have been computed for each study with computer programs written in Fortran and available on request.

In closing this section, let me agree with those who feel there has already been quite enough of surveying populations for gene frequencies in the vague hope that someday the information will be valuable (see discussion in *2*). In the case of the Indian, however, such surveys bear on a clearly defined problem on which considerable progress may be expected in the near future.

3. *The response of genetic systems to a rapidly changing environment.* As I mentioned earlier, we recognize that our species is all over the world subjected to a rapidly changing environment. Unfortunately, only in the last few years have biology and medicine found themselves in a position to begin to document with any precision what biological adjustments really take place. We have probably lost forever the opportunity to study what occurred in Caucasian populations. We are about to lose the opportunity to document what happens in Negro and Mongoloid (including Indian) populations.

The transition of surviving primitive Negro or Indian groups will be telescoped into a far shorter time period than was true for Caucasian groups. Thus, some Indian groups will be called upon to move from the Stone Age to the Atomic Age in a few hundred years, whereas the transition for most Caucasian groups took thousands. It seems reasonable to argue that here is an opportunity to study changes in population structure and adjustments to new stresses that we must not lose.

As for the previous two sections, so here the intellectual challenge is to convert an easily enunciated but somewhat diffuse series of problems into specific issues susceptible of precise study. A number of exact studies of population structure in transitional groups have already been completed or are in progress, notably those of Salzano and colleagues on the Caingang of Brazil (*19, 20*), of Layrisse and colleagues on the Warao of Venezuela, of Covarrubias and colleagues on the Pewenche of Chile, and of Cann and colleagues on certain Quiché speakers of Guatamala. There are also studies in the United States on groups in a more advanced stage of the transition, namely by Spuhler and colleagues on the Navajo (*21*) and Hackenberg on the Papago (*5*).

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[1] See Appendix of this volume.

Also in progress are studies of specific medical issues that arise in transitional groups, as the latter portion of this session will attest. The work of Roche and colleagues in particular will illustrate just how rapid and subtle may be the changes introduced by minimal acculturation. If genetic factors are important in the susceptibility to the thyroid hyperplasia and diabetes mellitus now so common in certain Indian groups, then, because of the recency, frequency, and intensity of the phenomena, these groups may be unusually favorable material for the study of these genetic factors.

Some unpublished studies of Drs. Warren Eveland, William Oliver, and myself may help point to some of the nuances of this transition. Six *E. coli* strains were isolated by Dr. Eveland from each of 77 Yanomama stools, each stool from a different subject. An effort is in progress to type each of the resulting 462 strains, using some 140 different typing sera. Thus far approximately 44 per cent of the strains fail to type out. By contrast, experience indicates that only a very small fraction of *E. coli* strains isolated in the United States would fail to type. What remains to be determined is whether this is a unique finding in a long-isolated population or whether similar studies on other such groups will reveal the same pattern, with the inference of a predictable shift in the intestinal flora with acculturation.

Some moral issues

It has seemed appropriate, in a presentation to an audience of scientists, to stress research opportunities. But as in the 1960's we increasingly recognize the issues created by scientific inquiry divorced from ethical and humanitarian considerations, it also seems appropriate to consider briefly what these studies, and especially the type mentioned in the last section, might mean to the Indian. We have no accurate census of the relatively pure Indians left in the Americas, or of the persons of mixed but "substantial" Indian ancestry. Estimates of the order of 16,000,000 (*17, 20, 24*) have been made for the former, while the latter come to easily several times that figure. We are talking about large numbers of people.

Who among us can read the history of the relations between the early settlers of his country and the Indians without deep shame for the barbarism heaped upon a people who were driven to defend the land they occupied? The world is watching my country as it agonizes over the Negro problem—it might equally well be watching the Americas for signs of a belated moral resurgence with respect to the Indians. How satisfied are any of us with the official programs of our governments for the health, economic advancement, and education of the Indians? How can we translate the results of our scientific investigations into concrete action programs, programs that must be carefully related to other government measures? It is a dubious favor to lower infant mortality among the Indians without parallel economic measures to ensure food for the extra mouths. Nor does it seem likely that Indians' accident-proneness (see references in *23*), so easy to attribute uncritically to their heritage of violence and lack of familiarity with our gadgets, will yield to education until the frustrations that contribute to accident-proneness are relieved.

In a world that seems to be groping for perspective, the Indian provides a reference point from which to view the fantastic disruptions that modern men, intrinsically still little different in all essential biological attributes from

Indians, have brought about. There are those who will take umbrage at my characterization of us representatives of Western culture as "intrinsically still . . . Indians." I am aware of the so-called intelligence tests that purport to show the inferior intellectual qualities of the American Indian, just as I am aware of similar results with the American Negro. These results can be and have been used as an excuse for less-then-equal schooling. But in both instances it is a matter of a culturally deprived and alienated group—perhaps also subjected to early nutritional deficiencies, whose role in impaired intellectual performance we are just beginning to recognize—being judged by tests designed by and standardized on a very different group (see also *4*). By these remarks I do not mean to dismiss the possibility of intellectual differences between ethnic groups, but only to insist that to date the data are grossly inadequate and we who call ourselves scientists must adhere to the null hypothesis, the more so since its various alternatives can be so conveniently misused by those who would evade their social responsibilities.

Be this last digression as it may, each time I return from the field there is a period of culture shock as I realize how greatly in a short period of time we have contrived to disrupt our ecosystem, and how profound is our ignorance of the long-range results of this disruption. Now in this time of greatly intensified concern over these problems, studies in depth of the Indian, within or without the framework of IBP, will surely contribute not only to his well-being but also to our own perspective and, eventually, the necessary adjustments toward which we are evolving.

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DISCUSSION

Dubos: Before we open the general discussion, Dr. Neel, I should like to ask you to take over again the problem raised by Dr. Waterlow a while ago. He asked about the evidence for the genetic character of some of the differences in blood groups and you answered. But I should like to ask if you could outline for us what kind of genetic evidence you can hope to obtain with regard to those adaptive processes of which you have spoken, especially with regard to the problems of susceptibility to disease, which will be discussed this afternoon. In other words, how possible is it in practice to obtain the kind of information that would determine whether the change is genetic or simply phenotypic?

Moderator: I am afraid that an answer to that question might anticipate a good deal of our discussion for this afternoon. It is relatively simple to obtain evidence for the genetic nature of the kinds of traits that Dr. Layrisse, Dr. Arends, and Dr. Lisker have presented. It is clearly much more difficult to obtain evidence for the genetic basis of such attributes as disease resistance.

We shall hear from Dr. Nutels this afternoon about some of the medical problems of Indian groups in transition. We have recently had the experience of being in the middle of a measles epidemic in a virgin population of Indians. It was a very instructive and sobering experience. But one of the strong impressions we came away with—and Dr. Centerwall will present some of the data—is that there may have been a great deal of uncritical thinking in the past about the constitutional susceptibility of the Indian to this, that, and the other disease. We feel that the secondary epidemiological characteristics of populations living at this level may determine the high mortality to a much greater extent than any innate susceptibility.

Now, how to pin down that kind of impression? It is very difficult. One practical approach is simply to provide the Indian, when he first encounters tuberculosis or measles or pertussis, with the same kind of medical care and nutrition that corresponding Caucasian populations are given and see what the difference is.

As you know, Dr. Dubos, there is a considerable literature, based largely on studies in twins, on disease susceptibilities in man. There is also a large literature of studies in experimental animals, the results of which are sometimes transferred to man. This literature leaves much to be desired; there is a need for precise investigations, as with standardized challenges under controlled conditions.

Waterlow: What is worrying me, Dr. Neel—and I am very ignorant of this subject—is that the markers you are studying may in a sense be of no physiological importance, just as the color of one's iris might be said to be of no importance physiologically.

I am assuming that the variety of polymorphic proteins, the isoenzymes such as the phosphatases, dehydrogenases, and so on, are also genetically determined. I do not know whether this is proved yet. Such proteins are likely on physiological grounds to be more important in relation to adaptability. What I am not clear about is how far your program of the kind of things you study is complete in itself. It rather looks as if geneticists had their own list of proteins that they call markers, whereas we biochemists are thinking of rather different types of proteins.

Moderator: Dr. Waterlow, you have asked a very perceptive question, on which, again, we could spend a great deal of time. Probably the most important development in human genetics in the past decade has been the realization of the tremendous amount of genetically determined biochemical diversity in our species. I, for one, think that one of the outstanding challenges in human genetics is to understand the significance of this diversity. What is it all there for? It is so great that we are having difficulty in seeing how it can all be maintained at once by selective pressures. The problem is to sort out which of these polymorphisms are like vestigial organs and which are meaningful to the species.

Now, to come more directly to how we get at this. It is quite clear that those polymorphisms arose in primitive man. And we feel that many clues to their function may come from primitive man. One of the things we attempt to do in our field work is collect all our data in family relationships. We can then analyze family material to see whether certain polymorphisms are associated with increased fertility or with decreased infant mortality. This is a long, slow business. Parallel studies on civilized populations are also needed. The numbers necessary to establish a significant association are tremendous. And, in fact, we almost have to think in a higher order of magnitude than our past studies when we try to come to grips with these issues.

To sum up, although we are interested in these traits as "markers," for which purpose we need not understand their physiological significance, we are also just as interested in understanding what they do.

Dubos: Do you see any possibility of determining whether adaptation to high altitude is a genetically determined or a purely phenotypic adaptation? I think this is relevant to our discussion last year.

Moderator: There is a great deal of interest now in adaptation to high altitude, not only in the Andes but also in the Himalayas and other parts of the world. And there is, as you know, a running debate on how much of the adaptation we see—for example, the barrel chest of the Andean Indian—is genetic and how much is simply physiological adjustment to life at high altitudes.

Now, one of the better opportunities to get at a question like that exists in Ethiopia. In the highlands and lowlands of Ethiopia are groups who on the basis of the genetic markers we have been discussing appear to be very similar. And yet one group lives at a very considerable altitude and one group not far above sea level. There is thus an experiment of nature, as it were, in which a group has been subdivided and the resulting subgroups placed in diverse environments.

The situation does not seem to be quite so simple in the Americas. We have not yet found the "control" for Andean man. Once there are such controls it is possible to investigate whether they are physically more similar to high-altitude man than other Indian groups—that is, whether there has been self-selection of groups able to live at high altitudes. There is also the hope of finding marital exchanges between the high- and low-altitude groups.

Dubos: Would you elaborate on the statements that were made last year about the anatomical differences in the heart between man born at a high altitude and man born at our altitude?

Moderator: I would prefer that those who made the statements were here to elaborate on them. As I recall it, what we were shown was evidence that changes appear very early on in the heart and lungs. But I did not see any convincing evidence that these early changes were genetic in nature. As far as I am concerned, it is a wide-open question whether this is genetic or an environmental adaptation.

Griffin: I have a suggestion on the general problem Dr. Neel is working on. There was a somewhat comparable dispersal, by a quite different group, into Australia at about the same time, according to radiocarbon dates. Studies of the Australian aborigines, which were a much less diversified and much smaller population, might be important with regard to evolution among "isolated" human groups.

Moderator: Yes, I agree that the Australian aborigine does represent the group most nearly comparable to the Indian. However, the disruption and decimation of the Australian aborigine is even further along than that of the American Indian. It is extremely difficult now to find groups of any size that have not been seriously disturbed by being displaced into very unfavorable country or in which there has not been a forced amalgamation of previously separate groups.

Cohen: Following up Dr. Waterlow's and Dr. Neel's remarks, I would like to ask Dr. Neel whether geneticists are being prepared for the fact that, at least in molecular biological or biochemical terms, the concept of the regulation of readouts at the translational level as well as at the transcriptional level is beginning to emerge. What brings this to mind is your allusion to the primitive Indians' often being relieved of the burden of contamination carried by our population. They are living with quite different intestinal flora, parasites, and perhaps even types of natural foodstuffs to which our population is not exposed, but I do not know whether in fact their environment can necessarily be spoken of as less contaminated. The primitive population would be less contaminated by synthetic and non-natural factors; relative to our own situation, however, it may be more contaminated. This comes to affect the whole question of separation of genetic potential because of the possibility that transcription may be regulated at the level of translation. There may be all kinds of factors in terms of the phenotypic result that could emerge by very subtle devices. I am wondering whether even the familial tests that the geneticists traditionally use would get around this problem.

Moderator: I would remind you that the geneticist has had quite a hand in molecular biology.

Obviously, there is at present no answer to your question about differences in transcription, qualitative or quantitative, at that cultural level as compared with our own. I think all of us who have done field work have emerged with a feeling that here in a very different world—and it is a very different world, the world of primitive man—you nevertheless quickly recognize all the familiar types of our own culture. You do not have to be very long in an Indian village before you get the same feeling for the strong men and the weak men and the operators and so on that you encounter in our own culture.

So I would say that at least part of the transcription is probably much the same at all cultural levels. Whether, on the other hand, there are subtle differences in the genetic readout, who can say?

McDermott: Along the lines of Dr. Dubos' question, Dr. Neel, are the people who are conducting the studies of lower-animal behavior also making these batteries of observations on the various genetic markers? I was thinking, for example, that the question of adaptability to high and low altitude might conceivably be approached from that standpoint. Is there work in this field, attempting to relate these genetic traits to the behavioral pattern of the animal?

Moderator: We know more about man's genetic constitution than about that of any other mammal at the present time. However, what might be called the comparative polymorphology of the primates is under very active investigation. A great deal of effort is now going into exploring the extent to which the higher primates have polymorphisms comparable to those of man. It is too early for any generalizations, but it is a fact that some polymorphisms encountered in man are found in the higher primates. On the other hand, the primates show polymorphisms that are not found in man. No pattern has yet emerged. I assume that in due time it will be possible to try again to make the kinds of associations you refer to. But, again, I come back to the problem of numbers. It is going to be very difficult to get large numbers on any of the higher primates. It is much easier with man.

Dubos: There can be no doubt that Dr. Neel has not only touched on but developed for us the foremost problems of man's adapting to the new conditions he is creating. All sorts of statements are continuously being made, such as "Modern man has become adapted to crowding" or ". . . cannot become adapted to crowding"; "Modern man has become adapted to pollution" or ". . . cannot adapt to pollution." And, in fact, these problems, which are so crucial to the whole development or survival of our civilization, have not as yet been formulated in sufficiently clear terms for us to determine whether we are dealing only with genetic adaptability, only with phenotypic characteristics, or whether we are simply playing on the polymorphic richness of the human race.

The problem is so large and so important that we obviously are not going to solve it here. But I hope at least that Dr. Neel will conclude with a general statement on this issue.

Moderator: I merely want to agree that we are not going to solve the problem this morning and to agree with one other thing Dr. Dubos has implied—that there are great difficulties in clear formulations in this field. How do we set up specific questions to which we can get what would be called scientifically hard answers? I think we are in accord that this is an important area. And I think we are in accord with respect to the methodology: we have a long way to go.

SURVEY OF THE UNACCULTURATED INDIANS OF CENTRAL AND SOUTH AMERICA[1]

Francisco M. Salzano

Acculturation and biological change

Before trying to make the survey mentioned in the title it is important to indicate the problems that arise when an attempt is made to define a specific group as "acculturated" or "unacculturated." The term *acculturation* is generally employed by American anthropologists, while their British colleagues prefer the word *culture contact* and the Cuban scholar F. Ortiz the term *transculturation,* which emphasizes the reciprocal character of most contact situations. Though much criticized and modified, the most widely used definition is that of Redfield, Lindon, and Herskovits (*19*): "Acculturation comprehends those phenomena which result when groups of individuals having different cultures come into continuous first-hand contact, with subsequent changes in the original cultural patterns of either or both groups."

A number of basic problems exist in the interpretation and analysis of these situations; they are reviewed by, among others, Beals (*1*) and Oliveira (*16*). For instance, we have sometimes conflicting views of culturalists and sociologists, and in some specific cases the intrinsic nature of the process and its consequences are difficult to identify. A new dimension, which so far as I am aware has not been properly analyzed in this context, is that related to the biological changes that occur and sometimes are decisive for the direction and speed of acculturative modifications.

Ribeiro (*20*) recognizes for Brazilian Indians three stages on the road to acculturation. The first would be represented by the isolated tribes —those living in regions not yet occupied by the Brazilian society and having only accidental and rare contacts with our civilization. Some simply avoid such contacts, while those living near pioneer settlements may have suffered violence and be frankly hostile to "foreign" people. In either case, they can obtain all they need from the region where they live and from their work; they are therefore able to maintain complete cultural autonomy. The second stage would be that of populations having intermittent contacts with civilization. They live in regions already reached by the expanding frontiers of the national society; though they maintain a certain cultural autonomy, they already have needs that can only be satisfied through economic relations with civilized people. The third stage is represented by groups in permanent contact with more numerous and more differentiated members of the civilization. They have lost their cultural autonomy to a large degree, being entirely dependent on others for the supply of many tools and products. They still preserve some of their traditional customs, but already show profound changes due to ecologic, economic, and cultural pressures.

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What concomitant modifications occur in the biology of these groups? Only a very limited start has been made on quantifying the observations made in groups at these three stages, and much more is needed to obtain a complete picture (*12, 13*). One important point that should be emphasized is the speed of these changes. Probably the first characteristics to be affected are the epidemiological ones, and here many groups that show all the other traits typical of untouched populations may present patterns resembling those of groups at other stages of acculturation. A typical example is provided by the Txukahamae, a Cayapó group of central Brazil (*14*). They have a completely autonomous life and in every instance are representative of an *isolated* population, but they already have a high incidence of tuberculin reactors. Since the Amerindian populations apparently had no contact with tubercle bacilli in pre-Columbian times, the question arises, From what groups did they get the infection? Their contacts with neo-Brazilians are recent and intermittent, and it is reasonable to suppose that the source of infection was the Gorotire, another Cayapó group from whom they separated at the beginning of this century but with whom they still have frequent interchange. The diffusion of this pathological agent in some ways parallels the diffusion of cultural traits. The speed of these epidemiological changes is such that it is improbable that the study of present groups, no matter how untouched, would give us exact duplicates of past populations. But investigation of the biological and nonbiological factors that influence the genetic and physiological adaptation of these groups to new diseases and infectious agents has intrinsic interest and can elucidate important problems of human biology.

Source of the data and their limitation

In the next section I shall try to present a survey of the relatively unacculturated Indians of Central and South America. Since I have first-hand contact only with Brazilian Indians, information concerning other countries has had to be limited to printed material and the personal communications of many colleagues. The rapidity of change in these populations and also the discovery of new groups indicate that such a list should be considered as merely an approximation of the situation in the several areas surveyed. Moreover, the publications examined sometimes do not give basic details about the degree of acculturation of the group in question. Even with these reservations, the list may perhaps serve to give a rough idea of the opportunities that still exist for the study of populations at this cultural level. In all cases the figures concerning the country's total and its Indian populations were checked in the most recent official publications. The references indicated are those examined in addition to these sources. Only relatively recent estimates were included; for reviews concerning the older ones, see Rosenblat (*21*) and Steward (*23*).

The survey

CENTRAL AMERICA

Political subdivisions: Bahamas, Belize, Bermuda, Costa Rica, Cuba, Dominican Republic, El Salvador, Guadeloupe (and dependencies), Guatemala, Haiti, Honduras, Martinique, Mexico, Netherlands Antilles, Nicaragua, Panama, Puerto Rico, Virgin Islands, West Indies

Total population: 77 million.

Countries with significant number of Indians: Belize, Costa Rica, El Salvador, Guatemala, Honduras, Mexico, Nicaragua, Panama.

Indian population: 8,000,000 (10 per cent of the area's total population).

Unacculturated Indians: Perhaps 1,000,000 (1.3 per cent of total population).

BELIZE

Total population: 100,000.

Indian population: 8,000–10,000.

Information about the Indians: Mainly Maya and Chekchi, who came from Mexico and Guatemala.

Unacculturated Indians: Probably none.

Reference: 9.

COSTA RICA

Total population: 1,500,000.

Indian population: 7,200.

Information about the Indians: Most important groups appear to be Bribrí (about 3,500), living in Talamanca Range in south, and Boruca (about 1,000) in southwest on the Pacific side. Remaining Indians belong to groups of Chorotegamangue (Nicoya Peninsula) and Guatuso (northern and western borders).

Unacculturated Indians: Probably none.

References: 9, 24.

EL SALVADOR

Total population: 3,000,000.

Indian population: 100,000.

Information about the Indians: Most important group is Pipil (about 80,000) of Nahuatl origin, concentrated in various towns in Sonsonate Department. Also smaller groups of Nahua and Cakchiquel distributed over several departments.

Unacculturated Indians: Probably none.

References: 9, 24.

GUATEMALA

Total population: 4,438,000 (1965).

Indian population: 2,500,000.

Information about the Indians: Main groups in center and west: Maya, Cakchiquel, Tzutujil, Uspanteca, Achí. West: Mam, Aguacateca, Jacaltec, Kanjobal, Chuj, Ixil. North: Kechkí, Pocomchí, Pocomam, Lacandón, Mopán. East: Chortí, Lacandón Chol, Chontal. Southeast: Xinca.

Unacculturated Indians: Some groups living in inaccessible places could provide information on the influence of a non-European culture on genetic and other traits.

References: 9, 18.

HONDURAS

Total population: 2,400,000.

Indian population: 110,000.

Information about the Indians: Main groups in west: Chorotega, Miquirano, Guajiro. On coast: Moreno. Other: Opatoro, Jicaque, Paya, Zambo, Miskito, Sumu. The Miskito have mixed with Negro groups.

Unacculturated Indians: Probably none.

References: 9, 18, 24.

MEXICO

Total population: 40,913,000 (1965).

Indian population: Estimates vary widely. A conservative number would be 5,000,000.

Information about the Indians: Main groups in north: Kikapú, Tepehuano, Nahuatl, Pame, Huastec, Otomí. Northern Pacific: Cora, Huichol, Yaqui, Seri, Papago. Center: Mazahua, Matlatzinca, Tarasco, Mazatec, Mixtec, Popoloco, Totonac, Zoque, some of previous groups. Southern Pacific: Chol, Tzeltal, Tzotzil, Tojolabal, Maya, Lacandón, Amuzgo, Tlapanec, Chatiño, Chinantec, Chocho, Chontal, Huave, Mixe, Triche, Zapotec.

Unacculturated Indians: Lacandón (only about 200). Some groups living in inaccessible places could provide information on influence of a non-European culture on genetic and other traits.

References: 4, 9, 18, 25.

NICARAGUA

Total population: 1,631,000 (1966).

Indian population: 80,000.

Information about the Indians: Most important groups are Miskito (with a large component of Negro mixture), Sumu, Rama, and Ulva.

Unacculturated Indians: Stone (*24*) mentions the Ulva tribe between the Mico and Siquia rivers, and the Sumu (3,000–4,000 in 1948).

References: 9, 18, 22, 24.

PANAMA

Total population: 1,314,460.

Indian population: 65,000–100,000.

Information about the Indians: Main tribes are Guaymi (about 36,000), Cuna (some 22,000), and Chocó (6,000). Two other small groups are Bocotá (200) and Teribe (400). Guaymi are found in Chiriquí, Bocas del Toro, and Veraguas provinces; Cuna mainly in San Blas Province, both mainland and some islands; Chocó in Darién Province.

Unacculturated Indians: Some Chocó groups of Panamanian-Colombian border (?).

References: 9, 24, 26.

SOUTH AMERICA

Political subdivision: Argentina, Bolivia, Brazil, Chile, Colombia, Ecuador, French Guiana, Guyana, Paraguay, Peru, Surinam, Uruguay, Venezuela.

Total population: 175 million.

Countries with significant number of Indians: All except Uruguay.

Indian population: 10 million (6 per cent of the area's total population).

Unacculturated Indians: Perhaps 980,000 (0.6 per cent of the total population).

ARGENTINA

Total population: 22,254,102 (1965).

Indian population: 130,000.

Information about the Indians: Main groups in Chaco region: Mataco, Chulupi, Chorote, Chiriguano, Pilagá (totaling about 30,000). Andean region: Quechua and Colla (some 18,000). South: Araucanian. Other regions: Cainguá, Mocobi, Toba.

Unacculturated Indians: Perhaps some Chaco groups.

References: 8, 9.

BOLIVIA

Total population: 3,800,000.

Indian population: 2,450,000.

Information about the Indians: Main groups in Andean region: Quechua (about 1,500,000) and Aymara (850,000). Northeastern region: The forest-dwelling populations (approximately 87,000) are divided into about 63 tribes belonging to 15 linguistic families. Some of them are as follows: Beni Department: Sirionó, Baure, Jora, Chacobo, Tacana, Chimane, Movima, Yari, Sirineire, Cayubaba, Itoma; Department of Santa Cruz: Guarayo, Borosese, Chamacoco, Zamuco, Lengua, Tapieti, Otuqui, Guatose, Yuracaré; Pando Department: Araona, Toronoma, Pacaguara, Machicanga; Department of La Paz: Chama, Chimane, Guarayo, Leco. Southeastern region (Bolivian Chaco): Tarija Department: Chiriguano, Mataco, Toba, Chulupi, Guaycuru, Chanese (about 14,500); Quiquisaca Department: Chorote, Chiriguano (some 3,800); Cochabamba Department: Sirionó, Yuracaré (2,000). General total for the region: approximately 50,000 Indians.

Unacculturated Indians: Almost all forest-dwellers.

References: 9, 28.

BRAZIL

Total population: 90,193,000.

Indian population: 100,000.

Information about the Indians: Tribes or groups classified by Ribeiro (20) and others as isolated or in intermittent contact (excluding those with fewer than 50 individuals): Agavotokueng, Apalai (100–250), Araras, Asurini (250–500), Atruahí, Barawâna, Boca Negra, Canoeiros, Cayabi (250–500), Diore (500–1,000), Gaviões (1,500–2,000), Guajá (100–250), Ipewi, Javai (250–500), Kabixí, Kalapalo (100–250), Kamayurá (100–250), Katawiân, Kren-Akoro (2,500–3,000), Kuben-Kran-Kegn (250–500), Kuben-Kragnotire (400), Kuikuro (100–250), Makú, Mandawáka, Maopityan, Makiritare or Mayongong (500–1,000), Mehináku (50–100), Mentuktire (500–1,000), Mudjetire (50–100), Nahukuá (50–100), Nambicuara (500–1,000), Pakaánovas (500–1,000), Parakanân (250–500), Parikotó, Pianokotó, (250–500), Puruborá (50–100), Salumá, Sikiâna, Tapayúna, Tirió (2,000–3,000), Trumãi, Txicáo, Txukahamae (200), Urubus-Kaapor (500–1,000), Waimirí, Waiwai (100–250), Waurá (100–250), Xavante (2,000–3,000), Xikrin (250–500), Yabaâna, Yanomama (2,000). For location, see references 11 and *19*. The former presents many other names, but it is not always clear whether tribes or tribal subdivisions are meant.

Unacculturated Indians: Perhaps about 40,000.

References: 11, 20.

CHILE

Total population: 8,567,000 (1965).

Indian population: 130,000.

Information about the Indians: Main groups in Northern region: Quechua and Aymara. Southern region: Araucanian (Mapuches).

Unacculturated Indians: Probably none.

References: 9.

COLOMBIA

Total population: 17,787,000.

Indian population: 300,000.

Information about the Indians: Main groups in Cauca region: Guambiano, Páez, Guanaco, Yanacona, Coconuco (approximately 80,000). Nariño: Quillacinga, Kwaiker, Inga, Kamsá (about 60,000). Guajira: Guajiro (50,000). Magdalena: Kogi, Ika, Chimila, Yuko (15,000). Antioquia: Cuna (2,000).

Unacculturated Indians: Perhaps 100,000.

References: 2, 9.

ECUADOR

Total population: 5,084,000.

Indian population: 2,000,000.

Information about the Indians: Main groups in coastal region and east: Colorado, Jívaro (13,000), Yumbo, Auca. Sierra: Quechua groups: Quindivana, Saguatoa, Salasate, Poato, Ilumane, Peguche, Quinchuqui, Quilotoa, Zumbagua, Pilaloe, Siquisilie, Puruhay, Cañaris, Saraguro.

Unacculturated Indians: Perhaps 250,000 (50,000 in the jungle).

References: 9, 15, 17, 28.

FRENCH GUIANA

Total population: 36,000.

Indian population: 1,200.

Information about the Indians: In coastal region: Galibi (600), Arawak (100), Palikour (100). In interior: Wayapi (130), Mereyo (60), Waiyana (200).

Unacculturated Indians: About 900 (Galibi, Palikour, Waiyana).

References: 3.

GUYANA

Total population: 647,000.

Indian population: 27,840.

Information about the Indians: Main groups in coastal region: Arawak, Carib, Warrau, Akawaio (about 10,000). In interior: Carib, Akawaio, Patamona, Arekuna, Makusi, Wapisiana, Waiwai.

Unacculturated Indians: Perhaps 2,000.

References: 3, Information note in America Indígena, 23: 158, 1963.

PARAGUAY

Total population: 2,030,000.

Indian population: 37,570.

Information about the Indians: They speak languages of 6 linguistic families and live in 17 locations. Main groups: Chiripá, Mbia, Tabïtërä, Guayaki, Guarayú, Chiriguano, Tapieti, Maká (about 600), Chulupi (more than 10,000), Chorote (approximately 100), Lengua (5,000), Sanapana (3,400), Guana (400), Angaite (400), Toba (1,400), Chamacoco, Morotoco.

Unacculturated Indians: Perhaps 10,000.

References: 9; data supplied by Department of Indian Affairs.

PERU

Total population: 11,800,000.

Indian population: 4,800,000.

Information about the Indians: Main groups in Northwest: Aguaruna, Jívaro, Huambisa, Andoa, Shapra, Curaraye, Chayhuita, Jebero. Northeast: Bora, Ocayna, Orejon, Pano, Yagua, Mayoruna, Chama, Huitoto, Ticuna, Cocoma, Capanahua, Cashibo, Shipibo. Center: Campa, Amuesha, Amahuaca, Marinahua, Cashinaua. Southeast: Sirinayri, Machigüenga, Piro, Mashco, Huarayo. Carneiro (*5*) also mentions following tribes from *montaña:* Yaminahua, Remo, Marobo and Iñapari.

Unacculturated Indians: Perhaps 450,000.

References: 5, 9, 27, 28.

SURINAM

Total population: 360,000.

Indian population: 4,500.

Information about the Indians: Main groups in coastal region: Galibi (2,400), Arawak (1,500). In interior: Waiyana (200), Trio or Tirió (400).

Unacculturated Indians: Perhaps 2,000.

Reference: 3.

VENEZUELA

Total population: 8,880,000.

Indian population: 62,700.

Information about the Indians: Layrisse and Wilbert (9) list the following: Guajiro (8,429), Paraujano (1,348), Yupa (2,057), Barí (1,000), Yaruro (1,427), Guahibo (5,397), Piaroa (1,886), Panare (412), Yabarana (64), Makiritare (1,200), Piapoco (99), Curipaco (212), Puinave (240), Bare (645), Yanomama (20,000), Sapé (100), Pemon (2,700), Warao (11,700), Cariña (2,776), unknown (1,000).

Unacculturated Indians: Perhaps 30,000.

References: 6, 7, 10, 29, 30.

Discussion

As was mentioned previously the estimates obtained present at least two types of error. One has to do with the identification of the acculturation process *per se;* the other is the proper placement of the group studied into "acculturated" or "unacculturated" categories. Because of the lack of detailed studies in most of the tribes surveyed here, and of reviews placing them in the appropriate category, the numbers obtained have a very relative value. They should be viewed as indicating orders of magnitude only.

With these reservations in mind it is now possible to examine the numbers arrived at. The population of Central America was estimated at 77 million and the number of unacculturated Indians at about one million (1.3 per cent). In South America the proportion is even lower: for a total of 175 million I calculate approximately 980,000 (0.6 per cent). But the fact that we may still have about two million largely "untouched" Indians points to the magnitude of the task, if we want to obtain truly comparative data in a number of populations living in different ecosystems. As a matter of fact, we simply do not have experienced persons to undertake the study of even, say, 10 per cent of this number in the immediate future.

With acculturation changes occurring so quickly, we need to work fast to establish on a firm basis the biological and nonbiological forces acting at this cultural level and determining the fate of these populations. This information is basic to our understanding of human evolution, but it is also important in relation to practical problems. The acculturation process is generally painful and distressing. In fact, a large proportion of the groups affected by it simply disappear physically. Data on populations living at different cultural levels can provide clues to an easier and more humane way toward technological advance.

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Appendix

ALPHABETICAL LIST OF TRIBES CITED[1]

Achí (Gua)
Agavotokueng (Br)
Aguacateca (Gua)
Aguaruna (Pe)
Akawaio (Gui)
Amahuaca (Pe)
Amuesha (Pe)
Amuzgo (M)
Andoa (Pe)
Angaite (Par)
Apalai (Br)
Araona (Bo)
Araras (Br)
Araucanian (A)
Araucanian (Mapuche) (Ch)
Arawak (Fr G, Guy, S)
Arekuna (Gui)
Asurini (Br)
Atruahí (Br)
Auca (E)
Aymara (Bo, Ch)

Barawâna (Br)
Bare (V)
Barí (V)
Baure (Bo)
Boca Negra (Br)
Bocotá (Pan)
Bora (Pe)
Borosese (Bo)
Boruca (CR)
Bribrí (CR)

Cainguá (A)
Cakchiquel (ES, Gua)
Campa (Pe)
Cañaris (E)
Canoeiros (Br)
Capanahua (Pe)
Carib (Guy)
Cariña (V)
Cashibo (Pe)
Cashinaua (Pe)
Cayabi (Br)
Cayubaba (Bo)
Chacobo (Bo)
Chama (Bo, Pe)
Chamacoco (Bo, Par)
Chanese (Bo)
Chatiño (M)
Chayhuita (Pe)
Chekchi (Be)
Chimane (Bo)
Chimila (Co)
Chinantec (M)
Chiriguano (A, Bo, Par)
Chiripá (Par)
Chocho (M)
Chocó (Pan)
Chol (M)
Chontal (Gua, M)
Chorote (A, Bo, Par)
Chorotega (H)
Chorotegamangue (CR)
Chortí (Gua)
Chulupi (A, Bo, Par)
Chuj (Gua)
Cocoma (Pe)
Coconuco (Co)
Colla (A)
Colorado (E)
Cora (M)
Cuna (Pan, Co)
Curaraye (Pe)
Curipaco (V)

Diore (Br)

Galibi (Fr G, S)
Gaviões (Br)
Guahibo (V)
Guajá (Br)
Guajiro (H, Co, V)
Guambiano (Co)
Guana (Par)
Guanaco (Co)
Guarayo (Bo)
Guarayú (Par)
Guatose (Bo)
Guatuso (CR)
Guayaki (Par)
Guaycuru (Bo)
Guaymi (Pan)
Huambisa (Pe)
Huarayo (Pe)
Huastec (M)
Huave (M)
Huichol (M)
Huitoto (Pe)

Ika (Co)
Ilumane (E)
Iñapari (Pe)
Inga (Co)
Ipewi (Br)
Itoma (Bo)
Ixil (Gua)

Jacaltec (Gua)
Javai (Br)
Jebero (Pe)
Jicaque (H)
Jívaro (E, Pe)
Jora (Bo)

Kabixí (Br)
Kalapalo (Br)
Kamayurá (Br)
Kamsá (Co)
Kanjobal (Gua)
Katawiân (Br)
Kechkí (Gua)
Kikapús (M)
Kogi (Co)
Kren-Akoro (Br)
Kuben-Kragnotire (Br)
Kuben-Kran-Kegn (Br)
Kuikuro (Br)
Kwaiker (Co)

Lacandón (Gua, M)
Lacandón Chol (Gua)
Leco (Bo)
Lengua (Bo, Par)

Machicanga (Bo)
Machigüenga (Pe)
Maká (Par)
Makiritare (V, Br)

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[1] Country abbreviations as follows:

A = Argentina
Be = Belize
Bo = Bolivia
Br = Brazil
Ch = Chile
Co = Colombia
CR = Costa Rica
E = Ecuador
ES = El Salvador
Fr G = French Guiana
Gua = Guatemala
Guy = Guyana
H = Honduras
M = México
N = Nicaragua
Pan = Panamá
Par = Paraguay
Pe = Peru
S = Surinam
V = Venezuela

Makú (Br)
Makusi (Guy)
Mam (Gua)
Mandawáka (Br)
Maopityan (Br)
Marinahua (Pe)
Marobo (Pe)
Mashco (Pe)
Mataco (A, Bo)
Matlatzinca (M)
Maya (Be, M, Gua)
Mayongong (Br)
Mayoruna (Pe)
Mazahua (M)
Mazatec (M)
Mbia (Par)
Mehináku (Br)
Mentuktire (Br)
Mereyo (Fr G)
Miquirano (H)
Miskito (H, N)
Mixé (M)
Mixtec (M)
Mocobi (A)
Mopán (Gua)
Moreno (H)
Morotoco (Par)
Movima (Bo)
Mudjetire (Br)

Nahua (ES)
Nahuakuá (Br)
Nahuatl (ES, M)
Nambicuara (Br)

Ocayna (Pe)
Opatoro (H)
Orejon (Pe)
Otomi (M)
Otuqui (Bo)

Pacaguara (Bo)
Páez (Co)
Pakaánovas (Br)
Palikour (Fr G)
Pame (M)
Panare (V)
Pano (Pe)
Papago (M)
Parakanân (Br)
Paraujano (V)
Parikotó (Br)
Patamona (Guy)
Paya (H)
Peguche (E)
Pemon (V)
Pianokotó (Br)
Piapoco (V)
Piaroa (V)
Pilagá (A)
Pilapoe (E)
Pipil (ES)
Piro (Pe)
Poato (E)
Pocomam (Gua)
Pocomchí (Gua)
Popoloco (M)
Puinave (V)
Puruborá (Br)
Puruhay (E)

Quechua (A, Bo, Ch, E)
Quillacinga (Co)
Quilotoa (E)
Quinchuqui (E)
Quindivana (E)

Rama (N)
Remo (Pe)

Saguatoa (E)
Salasate (E)
Salumá (Br)
Sanapana (Par)
Sapé (V)
Saraguro (E)
Seri (M)
Shapra (Pe)
Shipibo (Pe)
Sikiâna (Br)
Siquisilie (E)
Sirinayri (Pe)
Sirineire (Bo)
Sirionó (Bo)
Sumu (H, N)

Tabïtërä (Par)
Tacana (Bo)
Tapayúna (Br)
Tapieti (Bo, Par)
Tarasco (M)
Tepehuano (M)
Teribe (Pan)
Ticuna (Pe)
Tirió (Br)
Tlaponeco (M)
Toba (A, Bo, Par)
Tojolabal (M)
Toronoma (Bo)
Totonaco (M)
Triche (M)
Trio (or Tirió) (S)
Trumái (Br)
Txicão (Br)
Txukahamae (Br)
Tzeltal (M)
Tzotzil (M)
Tzutujil (Gua)

Ulva (N)
Urubus-Kaapor (Br)
Uspanteca (Gua)

Waimirí (Br)
Waiwai (Br, Guy)
Waiyana (Fr G, S)
Wapisiana (Guy)
Warao (V)
Warrau (Guy)
Waurá (Br)
Wayapi (Fr G)

Xavante (Br)
Xikrin (Br)
Xinca (Gua)

Yabaâna (Br)
Yabarana (V)
Yagua (Pe)
Yaminahua (Pe)
Yanacona (Co)
Yanomamö (Br, V)
Yaqui (M)
Yari (Bo)
Yaruro (V)
Yuko (Co)
Yumbo (E)
Yupa (V)
Yuracaré (Bo)

Zambo (H)
Zamuco (Bo)
Zapotec (M)
Zoque (M)
Zumbagua (E)

DISCUSSION

Moderator: Dr. Salzano, I wonder whether there is perhaps a decimal point misplaced. You estimate that there are 1,000,000 unacculturated Indians in Central America and 980,000 in all of South America. But when I go through your treatment of Central America country by country, I simply do not find those numbers. Where are these 1,000,000 concentrated?

Salzano: As I said, these are very preliminary estimates. They make allowance for the very large groups in Guatamala and Mexico on which we do not know enough for me to set a specific figure. But I am sure that there should be some groups there that are sufficiently unacculturated to be worth studying.

Cohen: How reliable is the tuberculin test in this population that you referred to in the case of central Brazil? Are you sure the Indians do not have histoplasmosis or some other condition that would cross-react and you would appear to be getting a tuberculin that may be unrelated to the exposure to the disease?

Salzano: Dr. Nutels will probably speak about that. But what I can say is that there are not only problems in relation to the nonspecific reaction but also problems of reading and the proper injection of the solution. We have just published a paper on these questions in *Tubercle*.

Moderator: To be a little more specific, we do know there is a high frequency of positive skin response to histoplasmin among many of the Indian groups of the interior, in the absence of any tuberculin reaction. So I do not think that in this particular instance cross-reactivity is a problem.

Cohen: I was using that only as an example. Unless one knows what the spectrum of sensitivity of a population is to a test that has been developed for a different population, one has some questions about the significance.

Hilleboe: I had the same question on this small point, and I wondered about two things. First, was anything done to see how much tuberculosis was really present in the group with positive tuberculin tests, and whether or not there was positive sputum? Second, in the group from which it was thought that the infection might have come, what was known about the amount of tuberculosis caused by mycobacterium tuberculosis and not atypical strains?

Moderator: That is just about to be discussed by Dr. Nutels.

MEDICAL PROBLEMS OF NEWLY CONTACTED INDIAN GROUPS

Noel Nutels

Before entering on the topic of this paper, I should like to present a few questions on the meaning of "newly contacted Indian groups": To what period of time should this classification be objectively applied? Might not a group classified as newly contacted have had direct or indirect contacts previously ignored? How do we define a tribe that has had one and only one rapid contact—for example, eighty years ago? How would we classify groups of Indians who, living in isolation in their own primitive environment, have had rare, intermittent, and brief contacts during the past sixty years? I shall not attempt to answer these questions. My aim in this paper is simply to mention facts and report on my personal experience, not to interpret them.

In considering the deficiencies of such a study it is necessary to take into account the difficulties that prevail in this enormous and primitive region, lacking in means of transportation and communication, where a dispersed and almost extinct population still live as in the Stone Age.

According to various authors cited by Wagley (20), the native population of Brazil at the time of the discovery is estimated at 1,500,000. Nowadays this population, scattered over the national territory, is reduced to approximately 80,000 Indians living in tribal conditions. Among the causes of this decline, infectious diseases—at times used deliberately as a means of extermination by so-called civilized men—have perhaps been the most efficient (*13*). Many are the documented instances of extermination of the indigenous population through grippe, measles, smallpox, venereal diseases, malaria, and other diseases (*15*).

In 1946, the Brazilian Government created the Xingu National Park in an area of 22,000 square kilometers in the upper basin of the Xingu River, one of the largest tributaries of the Amazon, at an altitude of 820 feet (see Figure 1). This region in the geographical heart of the country was considered by zoologists, botanists, anthropologists, and other specialists to be representative of primeval Brazil from the standpoint of flora, fauna, and human conditions. The present population is approximately 1,000 Indians distributed among the Kamaiurá, Kalapalo, Kuikuro, Waurá, Iawalapiti, Aueti, Trumai, Juruna, Aupatse, Nahukwá, Matipuhy, Kajabí (since 1952), Suiá (since 1960), Txukarramãi (since 1962), and Tchicão tribes (*5*). Table 1 shows the age distribution in the groups that have been examined.

These fifteen tribes are autonomous. Some groups now dispersed are to be found among them. There are others, not yet contacted, living in or around the Park. The tribes belong mainly to four linguistic groups: Tupian, Cariban, Arawakan, and Tapuyan. There is also an isolated linguistic group, the Trumai.

Access to the Park is almost exclusively by air, for there are no roads and river transport is poor. Only military and other government planes may land on the Park airstrips, and special permission is needed by all strangers wishing to enter. Communications within the Park are limited by the fact that it has only one airplane, a few canoes, and a radio station.

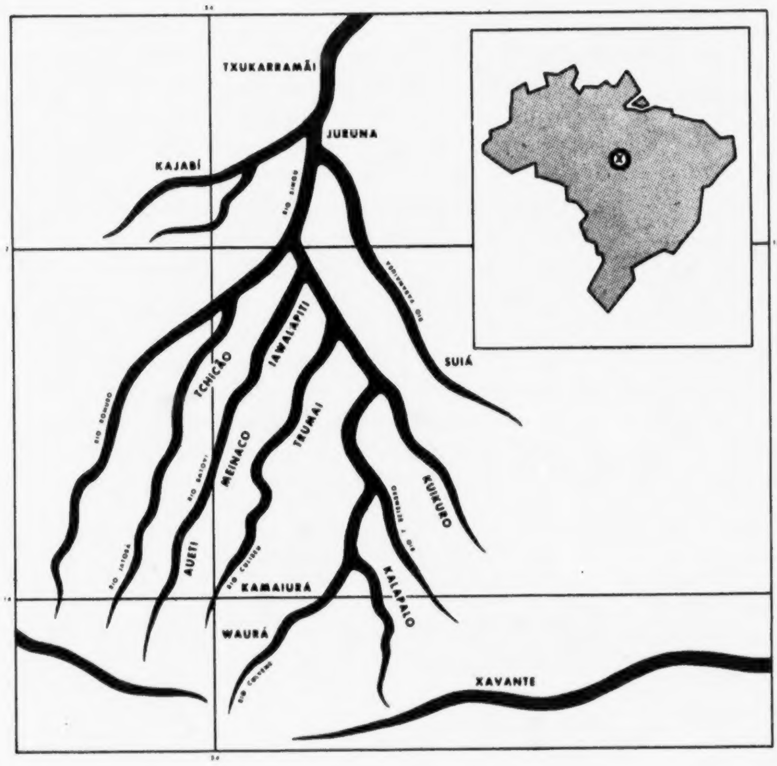
TXUKARRAMÃI
JURUNA
KAJABÍ
RIO XINGU
SUIÁ
TCHICÃO
IAWALAPITI
TRUMAI
MEINACO
KUIKURO
AUETI
KAMAIURÁ
KALAPALO
WAURÁ
XAVANTE

FIGURE 1. Location and distribution of tribes in Xingu National Park, Brazil. Inset shows location of tribes

Since its inception the Park has been directed by Orlando Villas Bôas, who is famous for his practical knowledge of the Brazilian backlands. Assisted by his brother Claudio, also well known in this field, he keeps a very close watch on the relations between the Indians of the area and the visitors, mainly anthropologists, ethnologists, psychologists, physicians, dentists, nurses, and others who do research in the Park or assist the Indians in various ways.

TABLE 1. Population of Xingu National Park, Brazil, in July 1967, by age group

TRIBE	NO. MEMBERS	AGE GROUPS 0–4	5–9	10–14	15–19	20–29	30 AND OVER
Kamaiurá	89	16	11	11	9	18	24
Kajabí	152	32	21	15	13	35	36
Kalapalo	68	14	14	5	11	13	11
Juruna	49	10	8	4	6	11	10
Meinaco	35	5	6	2	8	5	9
Suiá	67	11	7	11	6	18	14
Iawalapiti	34	2	8	3	3	6	12
Trumai	22	5	3	2	3	4	5
Aueti	40	9	8	6	—	3	14
Kuikuro	118	20	19	10	8	38	23
Waurá	62	17	14	1	7	15	8
Txukarramãi	170	36	21	19	16	33	45
Tchicão	53	9	4	5	9	22	4
Total	959	186	144	94	99	221	215

The first known contact between this area and so-called Western civilization occurred in 1884, when the first Karl von den Steinen expedition passed through (*18*). Three years later Von den Steinen came again. Expeditions since then include those of H. Meyer, in 1896; Max Schmidt, in 1900; the Rondon Commission, directed by Lieutenant Ramiro Noronha, in 1920; Percy Fawcett, also in 1920; Heinrich Hintermann, in 1924; the Rondon Commission, this time directed by Captain Vicente Vasconcelos, also in 1924; Percy Fawcett again, in 1925 (his second expedition, during which he disappeared); G. M. Doyott, in 1928; Vincent M. Petrullo, in 1931; and the National Service for the Protection of Indians, in 1940 (*1*).

In 1898 Karl E. Ranke, a German physician and demographer who had participated in the Meyer expedition, reported on the diseases he had observed (*12*). His account is still relevant, and I feel it is worth quoting in full:

> The diseases observed in 10 Indian villages among approximately 800 to 1000 Indians are the following: many healed fractures and one old haunch luxation but, according to the anamnesis, not congenital; one case of clubfoot, congenital; a very frequent skin disease, described as "Tinea imbricata," from the Malayan Archipelago and the Southern sea; numerous furuncles, most of them in the gluteal region; two cases of idiocy; one case of apparently parasitical tumor in the liver, which had as a consequence an ascites, not very developed; a kind of rheumatic disease of the joints; numerous cases of malaria and malarial "cachexia" in children under 10 years old; and a fairly mild enteritis among newborn infants. The occurrence of leucomas and staphylomas was extremely frequent in the Xingu area. Among the Bakairi from Kulisehu, none had been spared.
>
> With the help of a Bakairi from Paranatinga, the famous Antonio, who up to now has accompanied all expeditions to the Xingu area, I learned the history of their existence. Once, after the second expedition to the Xingu area, the Bakairi from Kulisehu went to Paranatinga and from there proceeded to Rosario. They were welcomed, and, although they had no knowledge of the Brazilian language, they were immediately baptized and after a few days' stay they went back carrying a heavy load of gifts.
>
> One of the tribesmen acquired an ophthalmic blennorrhagia in Rosario, which spread after his return to the Bakairi village on the Kulisehu and became a terrible epidemic. All the inhabitants were affected; some died, others survived with the loss of an eye or with leucomas. The numerous cases of conjunctivitis that I myself saw were all of a benign nature, so I think that the gonococcus has vanished from the Xingu area. It seems rather peculiar that I found no sign of it, not even congenital, and that it had not spread throughout the Indians or affected their sexual organs.
>
> Leprosy, syphilis, and tuberculosis are totally nonexistent in the Xingu area. The nonexistence of tuberculosis is of the greatest significance, since wherever Indians come into direct contact with white men, terrible devastation results.
>
> It may also be that measles, scarlet fever, and smallpox are likewise unknown in the Xingu area, although we are not so sure about their nonexistence at the time of our visit as we are in the case of the previously mentioned diseases. Only in the case of smallpox can we assure its nonexistence, because we found no one displaying smallpox scars.

In 1946 the white man took firm hold of the region, though not in large numbers. Before entering the area that later became the Xingu National Park, they were all given a medical examination. Between 1947 and 1950, in my capacity as a doctor in the Roncador-Xingu expedition, I imposed a sort of moral and sanitary quarantine on every person entering the region. Thus we were able to prevent venereal diseases and to protect the Indian women from the "civilized" men who would have competed with the native males.

At the time of their first contact with the Roncador-Xingu expedition of 1946, about 25 Kalapalo Indians died in an outbreak of grippe (*5*). Another outbreak was recorded in 1950 in the same tribe and also in the Kamayurá tribe, killing 12 persons this time. In June 1954 there was an epidemic of measles in the upper Xingu (*7*). Neither from the recollections of the elders nor from the traditions of any of the tribes were we able to gather information about it. Every Indian who came in contact with the Roncador-Xingu expedition at that time was struck by that outbreak. Of the 654 patients, 114 died. Among those who received medical care, the death rate was 9.6 per cent; among those who could not be treated in time, it reached 26.8 per cent.

Following is a list, on which I shall make no comment, of the diseases observed by us and other doctors who have been in the area: endemic malaria (*Anopheles darlingi, Plasmo-*

dium vivax and *P. falciparum*); *Tinea imbricata;* gastroenteritis; a purulent lung abscess of unknown etiology; furunculosis; childhood umbilical hernia; *arthritis deformans;* helminthiasis; *pemphigus foliaceus;* gallstones; *Blastomycosis cheloidiana* (*Paracoccidioides Loboi*) (*3*); warts; polymorphous acne; pediculosis; *Pityriasis alba;* gravidic striation; *Pulex penetrans;* conjunctivitis; pigmentary and cellular *nevi* (*6*); leucoma; pinguecula; pterygium; pupillary seclusion; melanic pigmentation of the conjunctiva; chalazion; cataract; ocular hypotension; talipes; malarial hematological disturbances (*17*); deformation of the auricular pinna due to a fighting game called *huca-huca;* atrophy of the lower limbs due to the ritual custom of prolonged constriction in infancy; scapulo-humeral luxation; tegumentary leishmaniasis (*4*); reversible paralysis of the lower limbs due to ritual ingestion of *mucunã* (*Dolichos pruriens* and/or *Dolichos urens*); toxoplasmosis, recently discovered (*2*); filariasis (*2*); and various arboviruses, recently discovered (*2*).

Since I believe that the purpose of my participation in this meeting is to tell about my personal experience in dealing with groups of recently contacted Indians, I shall concentrate on tuberculosis, which is the main field of my observations. Beginning in 1952, at first with X-ray and sometimes with old tuberculin (Von Pirquet modified: cutipuncture) and since 1960 with purified tuberculin (PPD-Rt 23-1 UT), I have investigated tuberculosis not only in the Park but also in other Indian villages, both those contacted long ago and those that have just been contacted (Table 2).

In 1952 the Kajabí Indians came to the Park from the Teles Pires River, in the valley of the Tapajós River, bringing with them *Blastomycosis cheloidiana* (Jorge Lôbo's disease) (*3*). This group had already been in contact with white men in its original area.

In 1960 the Villas Bôas brothers established contact with the Suiá tribe. This small group of approximately 80 Indians had had only sporadic encounters, over a three-day period, with the first Von den Steinen expedition in 1884. At that time Von den Steinen estimated their number at 150. No other encounter was recorded, even by Von den Steinen himself on his second expedition in 1887 (*19*). The presence of the Suiá group on the upper Paranajuba River was known because of their periodic attacks on other tribes, for which they were greaty feared. A few days after the Villas Bôas contact had been established, we were able to include 42 members of the tribe in our tuberculin survey (PPD-Rt 23-1 UT). Though at that time we did not have much suitable equipment and had had little experience with PPD,

TABLE 2. Reactions to tuberculin test (PPD-Rt23-1 UT) in Indian tribes of Xingu National Park, Brazil, July 1960

TRIBE	NO. TESTED	TESTS READ						
			0–4 MM		5–9 MM		10 MM +	
		TOTAL	NO.	%	NO.	%	NO.	%
Kamaiurá	76	76	74	97.4	2	2.6	—	—
Kajabi	63	63	60	95.2	1	1.6	2	3.2
Kalapalo	51	51	51	100.0	—	—	—	—
Juruna	45	45	45	100.0	—	—	—	—
Meinaco	38	38	36	94.7	—	—	2	5.3
Suiá	42	42	34	81.0	1	2.4	7	16.6
Iawalapiti	37	37	37	100.0	—	—	—	—
Trumai	17	17	17	100.0	—	—	—	—
Aueti	15	15	15	100.0	—	—	—	—
Kuikuro	3	3	3	100.0	—	—	—	—
Waurá	2	2	1	50.0	1	50.0	—	—
Total	389	389	373	95.9	5	1.3	11	2.8

we found eight positive reactions—one weak and seven strong—among this population, for an infection rate of 19 per cent. We also, on that occasion, gave the PPD test—again for the first time—to the other tribes of the upper Xingu, finding infection rates of 3.2 per cent among the Kajabí and 5.3 per cent among the Meinako. The former, as I have said, had had contact with civilized men before settling in the Park. The Meinako reactors, furthermore, had spent a few months in the city of São Paulo (*8*). The other tribes were found to be nonallergic. Subsequent research with X-rays revealed cases of lung shadows among the Suiá, and in 1967, for the first time, we were able to obtain from them a positive sputum by BK. In spite of all the natural difficulties, we were able to take a culture of bacilli in a Sula solution to the Central Tuberculosis Laboratory in the State of Guanabara, where they are being studied by Professor Milton Fontes Magarão and his staff (Tables 3, 4, and 5).

TABLE 3. Reactions to tuberculin test (PPD-Rt23-1 UT) among Suiá Indians of Xingu National Park, Brazil, June 1960

AGE GROUP	NO. TESTED	TESTS READ 0–4 MM NO.	%	5–9 MM NO.	%	10 MM + NO.	%
Adults	21	17	80.9	—	—	4	19.1
Children	21	17	80.9	1	4.8	3	14.3
Total	42	34	80.9	1	2.4	7	16.7

TABLE 4. Reactions to tuberculin test (PPD-Rt23-1 UT) by age group among Suiá Indians of Xingu National Park, Brazil, August 1966

AGE GROUP	NO. TESTED	TESTS READ 0–4 MM NO.	%	5–9 MM NO.	%	10 MM + NO.	%
0–4	8	8	100.0	—	—	—	—
5–9	10	9	90.0	—	—	1	10.0
10–14	13	11	84.6	—	—	2	15.4
15–19	5	4	80.0	—	—	1	20.0
20–29	22	17	77.3	1	4.5	4	18.2
30 and over	13	7	53.8	—	—	6	46.2
Total	71	56	78.9	1	1.4	14	19.7

In 1965, 180 Indians of the Txukarramãi tribe, fleeing the pressure of so-called pioneer fronts (in this case a group of *garimpeiros,* prospectors of precious stones and metal), sought refuge in the Park. These Indians, who belong to the Kayapó (Tapuyan) group, settled in a place called Porori, on the left bank of the Xingu River. When observed in 1962, this tribe had shown a low rate of tuberculin infection (Table 6). In 1966, after the Txukarramãi had already settled in the Park, we classified 158 of them in our thoracotuberculin file and examined their sputa (Table 7) (*10*). Three persons revealed BK in their sputa. Moreover, we discovered other strongly positive cases presenting radiologic forms of the type that do not usually reveal BK in direct sputum analysis, such as, for example, two ganglionary forms. All were treated with INH, PAS, and SM (Table 8).

In 1967 there came to the knowledge of the Park administrators the fact that a tribe of completely isolated Indians—the Tchikão—were being pressed by another group of prospectors. These Indians used to live on the banks of the Jatobá River, a subtributary of the Xingu. The few contacts they are known to have had consisted of attacks against other tribes living in the Park, from whom they attempted to take

TABLE 5. Reactions to tuberculin test (PPD-Rt23-1 UT) by age group among Suiá Indians of Xingu National Park, Brazil, July 1967

AGE GROUP	NO. TESTED	TESTS READ 0–4 MM NO.	%	5–9 MM NO.	%	10 MM + NO.	%
0–4	11	11	100.0	—	—	—	—
5–9	7	4	57.1	—	—	3	42.9
10–14	11	8	72.7	—	—	3	27.3
15–19	6	4	66.7	—	—	2	33.3
20–29	18	14	77.8	—	—	4	22.2
30 and over	14	6	42.9	—	—	8	57.1
Total	67	47	70.1	—	—	20	29.9

TABLE 6. Reactions to tuberculin test (PPD-Rt23-1 UT) among Txukarramãi Indians of Xingu National Park, Brazil, 1962

AGE GROUP	NO. TESTED	TESTS READ 0–4 MM NO.	TESTS READ 0–4 MM %	5–9 MM NO.	5–9 MM %	10 MM + NO.	10 MM + %
Adults	19	17	89.4	1	5.3	1	5.3
Children	20	20	100.0	—	—	—	—
Total	39	37	94.8	1	2.6	1	2.6

TABLE 8. Findings of tubreculin tests, X-rays, and bacilloscopy in Txukarramãi tribe of Xingu National Park, Brazil, August 1966

PULMONARY SHADOWS	TUBERCULIN TESTS 0–4 MM	TUBERCULIN TESTS 5–9 MM	TUBERCULIN TESTS 10 MM +	TOTAL
Minimum	—	—	1	1
Moderate	—	—	2(1*)	2
Advanced	1*	1	1*	3
Pulmonary Ganglion	1	1	2	4
Pleural	—	—	—	—
Total	2	2	6	10
No. X-rayed	105	11	32	148
Percentile of pulmonary shadows	1.9	18.2	18.7	6.7

* Positive sputum.

women and utensils. In order to prevent indiscriminate and undisciplined contact with members of our civilization, the administrators were able to resettle the whole 54-member tribe within the boundaries of the Park. We were then able to give these newcomers the tuberculin test at the moment of their arrival, with negative results.

In 1967 we repeated the research among the Txukarramãi and verified, this time, an absence of bacilli in the direct sputum analysis and the radiological regression of lung shadows in spite of an increase in the general rate of infection. This result was perhaps due to better equipment and technique employed in the test (Table 9).

We were surprised at both the clinico-radiological and the epidemiological aspects of tuberculosis in populations so primitive as the Suiá and the Txukarramãi. It was to be expected, once the disease was found among them, that an epidemic would have occurred that would have presented different forms such as the so-called infant form in adults and the rapidly evolving acute and miliary forms, similar to those found among the Senegalese soldiers taken to France during the First World War (*16*). Such dramatic epidemics among primitive peoples are often described in medical literature. Nevertheless, we were able to verify that among the Suiá and Txukarramãi the forms were the same as those that often occur among civilized white people, whose natural and acquired resistance gives them means to thwart the evolution of the disease. The X-rays presented here (Figure 2) leave no doubt, in my view, that

TABLE 7. Reactions to tuberculin test (PPD-Rt23-1 UT) by age group among Txukarramãi Indians of Xingu National Park, Brazil, August 1966.

AGE GROUP	NO. TESTED	TESTS READ 0–4 MM NO.	TESTS READ 0–4 MM %	5–9 MM NO.	5–9 MM %	10 MM + NO.	10 MM + %
0–4	36	31	86.1	1	2.8	4	11.1
5–9	25	19	76.0	2	8.0	4	16.0
10–14	15	10	66.7	2	13.3	3	20.0
15–19	16	14	87.5	—	—	2	12.5
20–29	28	18	64.3	1	3.6	9	32.1
30 +	38	23	60.5	5	13.2	10	26.3
Total	158	115	72.8	11	7.0	32	20.2

TABLE 9. Reactions to tuberculin test (PPD-Rt23-1 UT) by age group among Txukarramãi Indians of Xingu National Park, Brazil, July 1967

AGE GROUP	NO. TESTED	TESTS READ 0–4 MM NO.	TESTS READ 0–4 MM %	5–9 MM NO.	5–9 MM %	10 MM + NO.	10 MM + %
0–4	34	32	94.0	1	3.0	1	3.0
5–9	20	13	65.0	1	5.0	6	30.0
10–14	19	13	68.4	1	5.3	5	26.3
15–19	16	12	75.0	—	—	4	25.0
20–29	33	16	48.5	1	3.0	16	48.5
30 +	44	26	59.1	2	4.5	16	36.4
Total	166	112	67.5	6	3.6	48	28.9

tuberculosis among these Indians, in its clinical, radiological, and even epidemiological aspects, can be equated with that of peoples with a long experience with BK.

Had these tribes at one time undergone a so-called epidemic period of tuberculosis like other peoples? This question is not easy to answer. Is it possible that the BK responsible for the disease among them is less virulent than that responsible for the disease among whites? This hypothesis may be answered by the result of Professor Magarão's research mentioned above.

In any case, we believe that the Park is an appropriate environment for planned and controlled scientific research on the response of primitive populations to the introduction of tuberculosis and other infectious or noninfectious diseases by white invaders.

I consider it useful to conclude with Table 10, which shows the rate of tuberculosis infection among the Terena (*11*), Kayua, and Kadiweu Indians living in the southern part of Mato Grosso State, who have been in close contact with white civilization for at least a hunderd years (*9*).

TABLE 10. Reactions to tuberculin test (PPD-Rt23-1 UT) by age group among three tribes of Mato Grosso State, Brazil, September–October 1965

AGE GROUP	NO. TESTED	TESTS READ					
		0–4 MM		5–9 MM		10 MM +	
		NO.	%	NO.	%	NO.	%
0–7	584	559	95.7	4	0.7	21	3.6
8–14	634	537	84.7	37	5.8	60	9.5
15–49	746	525	70.4	71	9.5	150	20.1
50+	145	98	67.6	19	13.1	28	19.3
Total	2,109	1,719	81.5	131	6.2	259	12.3

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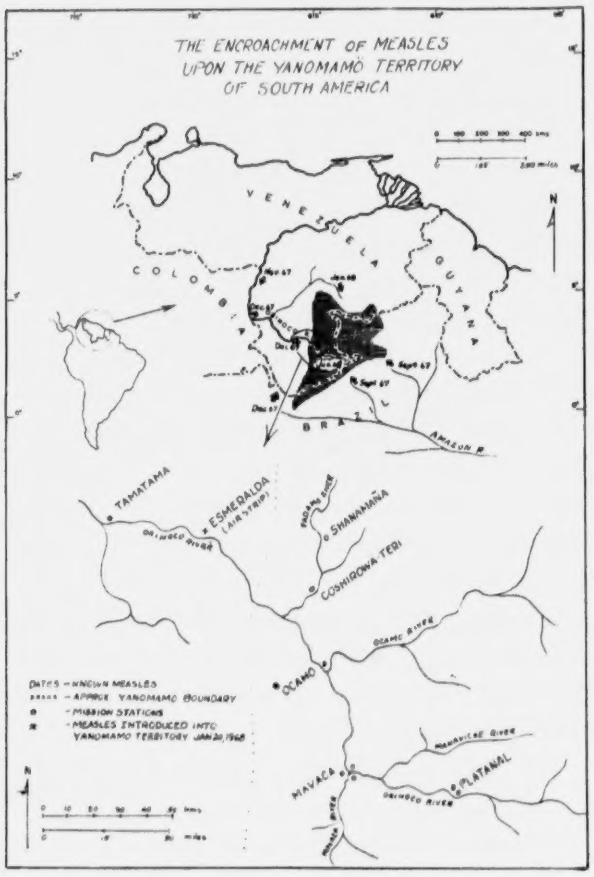
DISCUSSION

Willard R. Centerwall: A Recent Experience with Measles in a "Virgin-Soil" Population[1]

I have elected to use the time allotted for the presentation of recent experiences we have had on the effect of both measles and measles vaccine in a "virgin-soil" population of South America.

In January of this year a team of seven scientists, representing the United States and Venezuela, made an expedition into the Yanomama Indian territory. The map shows the approximate location of the territory, which covers an area extending from the Equator to 5 degrees north latitude, deep within the tropical rain forest of the Orinoco-Amazon River basin of Venezuela and Brazil. The expedition was oriented toward the collection of ethnographic, genetic, medical, dental, demographic, and epidemiological data. Three members of the team are present here today: Dr. James Neel, the expedition leader, who needs no introduction; Dr. Napoleon Chagnon, the anthropologist whose work, study, and life among these Indians since 1964[2] was so essential to our mission; and myself, a pediatrician with interest in and some experience with the diseases and nutritional disorders of the tropics. This was the third such expedition led by Dr. Neel into this general region, the first having taken place in 1966 on the Venezuelan side and the second in 1967 on the Brazilian side. The present expedition was in essence a cooperative venture of the University of Michigan and the Venezuelan Institute for Scientific Research, and in particular Drs. Layrisse and Tulio Arends of the Institute. Both are present at this session also; thus, if what I say in the next few minutes generates questions, there are several resources available for possible answers.


THE ENCROACHMENT OF MEASLES
UPON THE YANOMAMÖ TERRITORY
OF SOUTH AMERICA
VENEZUELA
COLOMBIA
GUYANA
BRAZIL
AMAZON R.
N
Nov 67
Jan 68
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Dec 67
Sept 67
Sept 67
Dec 67
TAMATAMA
ESMERALDA (AIR STRIP)
ORINOCO RIVER
PADAMO RIVER
SHANAMAÑA
COSHIROWA-TERI
OCAMO RIVER
OCAMO
MANAVICHE RIVER
MAVACA
PLATANAL
ORINOCO RIVER
MAVACA RIVER
DATES – KNOWN MEASLES
– APPROX. YANOMAMO BOUNDARY
– MISSION STATIONS
– MEASLES INTRODUCED INTO YANOMAMO TERRITORY JAN 20, 1968
0 10 20 30 40 50 kms
0 10 30 miles
0 100 200 300 400 kms
0 100 200 miles

There are believed to be about 10,000 Yanomama Indians living in more than 100 villages scattered throughout the region. They are very primitive forest-dwellers who practice a slash-

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[1] Investigations supported in part by U.S. Atomic Energy Commission Grant AT (11-1)–1552.

[2] Chagnon, N. A. Yanomamo Warfare, Social Organization and Marriage Alliances. Ph.D. thesis, The University of Michigan (1966).

and-burn agriculture along with hunting and gathering. Although some mission stations have been established along the Upper Orinoco and its tributaries since early in the past decade, the majority of these Indians are as yet uncontacted and unaffected by the outside world.

The typical Yanomama village is a circular stockade, with families living in hammocks under the shelter of the enclosure. In general the people are healthy and handsome, well proportioned and strong but almost pigmoid in stature. Their cheerful, friendly countenances bely the aggressiveness and hostility for which they are sometimes known.

Because measles antibody studies in 1966 and 1967 demonstrated that this was a virgin-soil tribe and because measles was known to have penetrated into the nearby regions, practically surrounding the Yanomama territory by 1967 (see map), we brought along with us into South America 2,000 doses of donated Edmonston-strain attenuated live measles vaccine, plus gamma globulin.[3] Half of these doses we diverted to the mission stations on the Brazilian side, where measles was beginning to penetrate the region, and the remainder went with us. We had intended to vaccinate and to advise the missionaries on the procedure toward the end of our scientific studies. We did not anticipate encountering measles during our two-to-three-month expedition. It was thus a fortuitous occurrence that our arrival into the region was almost simultaneous with the introduction of measles (we believe for perhaps the first time) into this territory.

A party of Brazilian workers had, the week before, come across from the Rio Negro region in Brazil to the south. They were at the Ocamo mission, at the junction of the Orinoco and Ocamo rivers, when a 14-year-old boy among them became sick with a high prostrating fever. He was seen shortly after the onset by Dr. Marcel Roche, Director of the Venezuelan Institute for Scientific Research, who happened to be at the mission in connection with a study on thyroid physiology.

The boy never developed the characteristic morbilliform rash, so the differential diagnosis from any of a variety of "jungle fevers" was uncertain; nevertheless, 31 Indians and 9 Brazilians at the mission were vaccinated at once with no gamma globulin coverage. It turned out that the boy indeed did have measles and that visitors to the mission meanwhile had returned to their own villages carrying the virus in its incubating form with them. Thus started a full-scale epidemic, which, by the time our last team member had left the field three months later, had spread up and down river along a hundred miles of waterways and tributaries and adjacent inland regions involving no fewer than 15 villages.

Within a few weeks we had exhausted our vaccine supply. Observations on these Yanomama Indians of the effect of the wild measles and of the measles vaccine with and without the concomitant gamma globulin form the basis of this report. Since it was neither conceived nor designed as a scientific study in itself and since our data-collection responsibilities did not permit a prolonged stay in any one village, our observations are of necessity disjointed and fragmentary, but still, we believe, of sufficient interest and value to warrant presentation.

Most of the Indians and also the several Brazilians we observed became very ill and prostrate with their measles. In several Indian villages where documentation was possible by direct examination, from 24 to 43 per cent of the victims had frank bronchopneumonia, with onset usually during the acute attack and sometimes during the recovery phase. This seemed to respond well to antibiotic therapy. We were unable to evaluate the possibly adverse effect of a respiratory flu-like epidemic, which had immediately preceded and sometimes co-existed with the measles epidemic. The complication of otitis media appeared to be practically nonexistent.

We were particularly impressed with the devastating effect of measles in its near-total involvement of the Indian community. When parents and children were simultaneously involved, there was a drastic breakdown of both the will and the means for necessary nursing care. We have even seen several instances where three generations of Indians were simultaneously ill with measles.

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[3] We are most grateful to Parke, Davis and Co., Phillips Roxane Laboratories, and Lederle Laboratories for their donation of the vaccine.

The reaction of those not already prostrated in a fatalistic depression was usually one of panic. Sometimes the well members of the village even abandoned the sick, and normal community structure and function were lost. Village fragments often fled to other villages for haven, thus increasing the spread of the disease.

In three villages with approximately 170 cases of measles there were 29 deaths, a rate of 17.1 per cent. Most of these were due to associated pneumonia, complicated by dehydration. This despite some measure of applied, though less than optimum, nursing care. In a fourth village, the authors were present for two days at the peak period of measles reaction involving nearly 40 Indians, a majority of them extremely ill. Vigorous and dramatic treatment included fluid administration, cooling measures, aspirin, and long-acting antibiotics. No deaths occurred during our stay, but we did hear later that subsequently two Indians died (approximately 4 per cent mortality).

These and other observations supported our feeling that differences in medical and nursing care play the major role in the measles-mortality rate among the Indians. In this connection, it should be recalled that the mortality from measles among Caucasians in the United States only half a century ago was in excess of 3 per cent; it has steadily dropped to a present-day low of less than 0.1 per cent. Nearly two thirds of this reduction occurred in the period before the advent of modern chemotherapy and antibiotics.[4] Other evidence that the death rate in highly susceptible groups can be substantially reduced by elementary organization and medical care emerges from a 1954 study on a measles epidemic among the Eskimos of the Canadian Arctic[5]; in the first of two outbreaks the death rate was 7 per cent, whereas in the second, with better organization and provision of medical care, it was only 2 per cent.

The use of measles vaccine permitted us to see some of its effects on the Indians. In villages where we could observe and measure the effects at their height, we found no real complications but considerable reaction, especially in terms of febrile response. That this response was associated with the production of protective measles antibodies was borne out clinically. To our knowledge, no persons vaccinated prior to the exposure to measles later developed the disease. This protective value of the vaccine showed up in peculiar ways, as for example in the case of an unvaccinated mother who became very ill with measles while her previously vaccinated and healthy youngster was playing about and pestering her for attention she could not give. This is just the reverse of the situation commonly seen in civilized societies.

Searching the literature for reports of reactions of other peoples to similar vaccine both with and without concomitant use of gamma globulin, we observed that these Indians in general seem to react with a greater febrile response than has been reported in North American Caucasians,[6] Europeans,[7] Icelanders,[8] Africans[7, 9] and Micronesians.[10] Again, however, as with the wild measles, we are unable to define accurately the role of concurrent respiratory infections in enhancing the reaction to the vaccine.

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[4] Langmuir, A. D. Medical importance of measles. *Am. J. Dis. Child.* 103:224–226, 1962.

[5] Pert, H. F. W., and F. P. Nagler. Measles in the Canadian Arctic, 1952. *Canad. J. Pub. Health* 45:146–156, 1954.

[6] Krugman, S., J. P. Giles, A. M. Jacobs, and H. Friedman. Studies with live attenuated measles virus vaccine. *Am. J. Dis. Child.* 103:353–363, 1962.

[7] Cockburn, W. C., J. Pecenka, and T. Sundaresan. WHO-supported comparative studies of attenuated live measles virus vaccines. *Bull. Wld. Hlth. Org.* 34:223–231, 1966.

[8] Gudnadotter, M., and F. L. Black. Response of adults in Iceland to live attenuated measles vaccine. *Bull. Wld. Hlth. Org.* 30:753–762, 1964.

[9] Meyer, H. M., Jr., D. D. Hostetler, Jr., B. C. Bernheim, N. G. Rogers, P. Lambin, A. Chassary, and J. E. Smadel. Response of Volta children to live attenuated measles virus vaccine. *Bull. Wld. Hlth. Org.* 30:760–781, 1964.

[10] Brown, P., M. Basnight, and D. C. Gajdusek. Response to live attenuated measles vaccine in susceptible island populations in Micronesia. *Am. J. Epidem.* 82: 115–122, 1965.

Summing up, although the evidence for genetic influences in mammalian resistance to a wide variety of diseases is of course incontrovertible, the present data nevertheless suggest that given a comparable previous disease experience (other than measles), similar care when ill, and a less resigned attitude toward the prospect of death, the death rate from measles in a previously unexposed Indian population would not be much greater than in a group of "virgin-soil" civilized Caucasians whose ancestors had been repeatedly exposed to the experience of measles. There is thus, in our opinion, only very limited evidence for a greater innate susceptibility of the Indian to this disease. It seems likely that a re-evaluation is in order with respect to the primary susceptibility of the Indian to certain other diseases of civilization, such as pertussis, smallpox, and tuberculosis.

General Discussion

Moderator: As I am sure you gathered from that capsule presentation, there were some problems in conducting ourselves like calm and detached observers in the midst of a measles epidemic. And when it was all over, we realized that in many respects we had failed to document this experience adequately.

Dr. Nutels in his presentation gave data on how the mortality rate in an Indian group can be lowered by even what I think he would agree is less than adequate medical care. Certainly this was our experience—that the mortality rate for Brazil and Venezuela in this epidemic would run under 10 per cent, which is a half to a third of what is often reported. It is very difficult to say how much lower this rate would go if these were Indians given the kind of care that a Caucasian would receive today in Europe or in the United States, but we believe it would drop down to a few per cent.

The other side of the coin, of course, is revealed by Caucasian populations that after centuries of exposure to measles have somehow become "virgin-soil" populations and have then experienced an epidemic of measles. The classic example is the epidemic Dr. Peter Panum reported from the Faroe Islands. A Danish population had been free of measles for some 65 years and then was stricken much as an Indian village is stricken.

I was most impressed by Dr. Nutels' remarks about tuberculosis and what I took to be his questioning attitude toward how differently the Indian reacts to it. Now, quite aside from its theoretical interest, this seems to me a matter of great practical importance. If we believe that the Indian is more susceptible to tuberculosis or measles or pertussis, we are apt to be satisfied with less than optimal clinical results in the management of those diseases. But if we think he is no more susceptible than we are, then we will not settle for anything less than the best. This is perhaps an important point that has been lost sight of in the past.

McDermott: I think both of these presentations represent very fine contributions to our accumulating knowledge on this subject. In previous meetings of this committee, several of us—among them Dr. Neel and I—expressed the belief that evidence that a genetic predisposition played a significant part in measles and tuberculosis should be very sharply challenged. Indeed, we did not believe it.

It seems to me the evidence in both these studies is in that direction. As Dr. Nutels said, it is conceivable that the strain of mycobacteria either may not have been tuberculosis or may be attenuated, but I do not think that is very likely. The evidence he presented makes it perfectly credible that these people did indeed have tuberculosis infection and disease. Our challenge to notions of genetic predisposition arises from our greater knowledge nowadays of how these two particular infections, measles and tuberculosis, are spread. Both can be spread (though this is not necessarily the only way) by what is known as the airborne route. When this is possible a whole constellation of circumstances having to do with the culture, and in particular the material culture and way of living, then obtains. So far as measles is concerned, we must remember first of all that it may be a very severe disease in any adult. Adults with measles are just as sick as the adults Dr. Centerwall was describing.

Secondly, the incidence of complications of measles—and the complications are what people die of—can be very high, depending upon the age at which the infection is acquired. Seventy-five per cent of the post-measles complications in New Delhi, for example, where they have large numbers, occur under the age of 18 months. In the particular circumstance of primitive societies with high sustained fertility, a high proportion of the population exposed is extremely young.

As for tuberculosis, I think the evidence is even clearer, because work over decades has proved the need to distinguish between tuberculosis *infection* and tuberculosis *disease*. It is only since the infection has become less prevalent that we have been focusing on it and hence have numbers on which to base greater understanding.

Tuberculosis infection can be spread and probably usually is spread by the airborne route. This means several things. One is that the infectious agent—in this case the tubercle bacilli—can be suspended in the air and in an enclosed space will remain in that air whether a human is there or not; anyone entering the room could acquire the infection. How long the air will stay poisoned is not known, but it is an appreciable interval—a matter of an hour or two. And the actual event of infection takes place presumably in a flash. This is relevant to contacts with previously unexposed populations.

In any case, the evidence now is quite convincing that given the proper physical circumstances for the transfer of tubercle bacilli from one person to another, circumstances such as are frequently seen in unventilated huts in aboriginal or primitive societies, there is no inherent immunity against infection with tubercle bacilli. By this I mean that practically all members of a group exposed under the proper circumstances, irrespective of their diet or way of life or level of civilization or anything else, will become infected. We know of course that infection leads to disease in only a minority of those infected; the exact percentage is not known. If we had, let us say, 10 per cent diseased in a society that was virtually 100 per cent infected, we might simply think that 10 per cent is an awful lot of tuberculosis and hence that this group of people, this race, is especially predisposed to it, whereas in point of fact they may have no more tuberculous disease *per infection* than any other group. It is simply that the particular circumstances of their society allow infection to be more prevalent.

Now with both tuberculosis and measles, and certainly with measles in infancy, there is of course an additional factor: nutrition. Malnutrition and lack of high-quality protein are characteristically present in the so-called unacculturated societies. What I am really saying is that our knowledge of the pathogenesis of both infections now gives us perfectly credible alternative explanations for what has been seen without its being necessary to invoke the idea of a significant hereditary predisposition.

Behar: How was the nutritional status of the children in that population determined?

Centerwall: By simple inspection and physical examination. The nutritional status of the children was remarkably good as a whole. There were a few areas in which there was active malaria, and some of the children were sick with that. But in general they were fine. There was no evidence of the protein deficiency seen in India and other tropical areas of the world.

Moderator: The next part of our program is devoted to some special medical problems affecting Indian populations.

THE PROBLEM OF GALLBLADDER DISEASE AMONG PIMA INDIANS

Thomas A. Burch, Leonard J. Comess, and Peter H. Bennett[1]

Introduction

The etiology of gallbladder disease is uncertain, though the problem has excited considerable speculation and controversy. Diet (*11*), female hormones (*10*), obesity (*16*), and diabetes (*13*) have all been said to be associated with cholelithiasis. In addition, differences in the frequency of gallbladder disease among various racial groups have been noted. On the basis of autopsy data Caucasians have been reported to have a higher frequency of gallstones than Negroes (*6*), and several studies based on hospital admission rates have suggested that American Indians have a still higher prevalence of gallbladder disease (*9, 12, 15*). A recent epidemiologic study (*5*) has confirmed the higher prevalence of "clinical gallbladder disease" among Pima Indians than was established by identical criteria among the predominantly Caucasian community of Framingham, Massachusetts (*7*) (Table 1).

The purpose of this paper is to report preliminary data on a partly completed epidemiologic study of gallbladder disease based on cholecystography in a population sample of Pima Indians, a Uto-Aztecan tribe living in the desert of the southwestern United States.

Methods and materials

Sample selection

Since 1963, the Clinical Field Studies Unit of the National Institute of Arthritis and Metabolic

TABLE 1. Prevalence of documented gallbladder disease in Framingham community and among Pima Indians

AGE AT ENTRY INTO STUDY (YEARS)	FRAMINGHAM GROUP			PIMA INDIANS		
	NO. AT RISK	WITH DISEASE		NO. AT RISK	WITH DISEASE	
		NO.	%		NO.	%
Males						
30–39	832	1	0.1	51	1	2.0
40–49	779	7	0.9	45	1	2.2
50–62	725	23	3.2	57	7	12.3 [a]
Total	2,336	31	1.3	153	9	5.9 [a]
Females						
30–39	1,037	24	2.3	45	16	35.6 [a]
40–49	963	58	6.0	51	20	39.2 [a]
50–62	873	88	10.1	62	21	33.8 [a]
Total	2,873	179	5.9	158	57	36.0 [a]

[a] $p < 0.01$.

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[1] Presented by Dr. Burch.

Diseases has been performing prospective studies of the natural history of arthritis (*2*) and diabetes mellitus (*14*) among Pima Indians living on the Gila River Indian Reservation in Arizona (see Figure 1).

A house-to-house census of the Gila River Reservation was compiled, using aerial survey photographs to ensure that all houses were visited. Attempts are made to examine each reservation Indian aged fifteen years and over every two years. This biennial examination includes a 75-gram oral glucose tolerance test; a history and physical examination pertaining primarily to diabetes and arthritis; radiographs of the chest, cervical spine, hands, feet, pelvis, thigh, and calf; a 12-lead electrocardiogram; photographs of both optic fundi; a bio-thesiometer examination (quantitative test of vibratory sensation in both great toes and index fingers); a urinalysis; and determinations of serum cholesterol, creatinine, uric acid, hematocrit, and rheumatoid factor by sheep-cell agglutination and bentonite flocculation titers. In addition, the blood of each subject examined has been typed for several blood groups.

A sample of 600 Pima Indians was selected with random numbers for a study of gallbladder disease. This sample consisted of 50 males and 50 females in each of the six decades between 15 and 74 years of age. Since the gallbladder study was started on 1 February 1967, 258 of the 600 members of the sample have attended the Unit for their comprehensive biennial examination. The subjects were selected according to their age at the time of the census (as of 1 January 1966), but in the present analyses they have been grouped according to their age at the time of the examination for gallbladder disease.

Cholecystogram procedure

Eight Telepaque [2] tablets were ingested on the evening prior to examination and the respondent was instructed to take nothing thereafter by mouth except water until the cholecystogram had been completed. Prior to the examination, each respondent verified that he or she had followed the instructions. No attempt was made to cleanse the bowel with laxatives prior to the cholecystography.

Initially two radiographs, a 14″ x 17″ PA scout film of the abdomen and a 10″ x 12″ oblique film of the right upper quadrant, were taken on each respondent. If the gallbladder was identified on either of these views, a 10″ x 12″ upright PA and a 10″ x 12″ right lateral decubitus view of the right upper quadrant were obtained. After this, most respondents were given a fatty meal consisty of 30 cc. of Cholestim [3] gallbladder stimulant and either the upright or the decubitus film was repeated.

Respondents in whom no gallbladder could be identified on the scout and oblique films were considered to have a nonvisualizing gallbladder, and an attempt was made to perform a repeat cholecystogram on them four to six weeks later.

Clinical gallbladder disease at surgery or by cholecystography had been documented previously in 60 of the 258 members of the sample who have attended the NIAMD Clinic since the beginning of the gallbladder study. An oral cholecystogram has been performed on an additional 132 of these respondents. The present report analyzes the findings on this subsample of 192 Pima Indians.

Of the remaining 66 respondents, 61 did not have a radiologic examination—8 because of a contraindication to the procedure, 43 because work or school made it impossible for them to return at the specified time, and 10 because they refused cholecystography. Five respondents were excluded because of the poor technical quality of their radiographs.

Criteria for Gallbladder Disease

The diagnosis of previously documented gallbladder disease was based on either a hospital record of gallbladder surgery, the patient's history of having had gallbladder surgery, or a hospital record of an abnormal cholecystogram (stones or a nonvisualizing gallbladder).

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[2] Iopanoic acid manufactured by Winthrop Laboratories, New York.

[3] Distributed by General Electric X-Ray Department, Milwaukee.

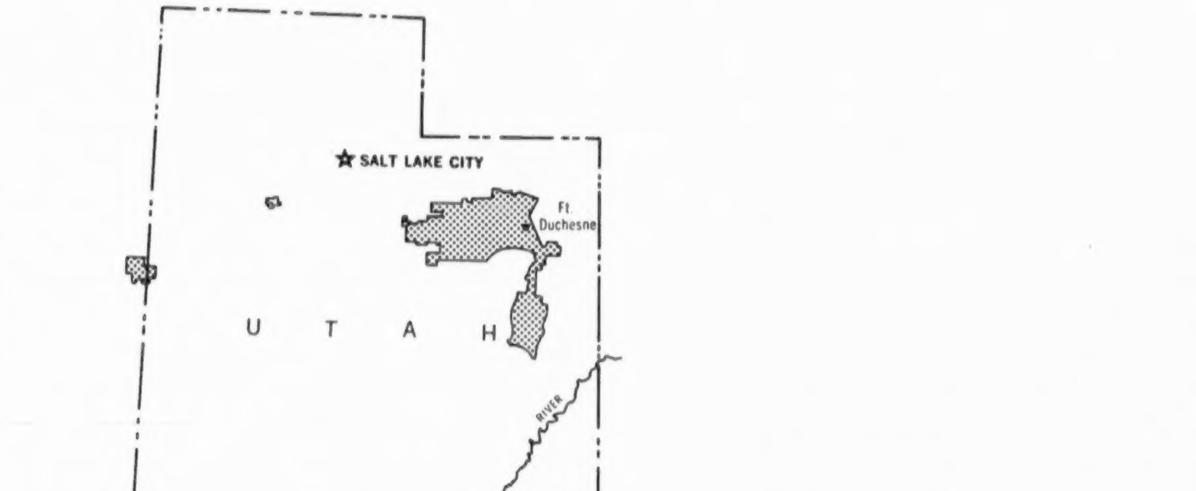
SALT LAKE CITY
Ft. Duchesne
U T A H
RIVER
COLORADO
NAVAJO
HOPI
LITTLE COLORADO
RIVER
COLORADO
RIVER
A R I Z O N A
N E W M E X I C O
SANTA FE
UNITED PUEBLOS
RIO
GRANDE
RIVER
FT. APACHE
RIVER
SAN CARLOS
SALT
PHOENIX
GILA RIVER
Sacaton
GILA
RIVER
GILA
RIVER
PAPAGO
Tucson

FIGURE 1. Indian reservations of southwestern United States. Gila River Reservation is at lower left.

The cholecystograms performed at the Clinical Field Studies Unit Clinic were analyzed for the patient's clinical record by a radiologist of the Division of Indian Health. All radiographs were reanalyzed by one of us (L. J. C.) prior to this report without knowledge of the age or sex of the respondents. A disagreement in interpretation was noted in only eight instances; these cases were carefully re-evaluated (by L. J. C.) without reference to the previous readings and the new interpretation was used.

Results

Prevalence of gallbladder disease

Table 2 shows that gallbladder disease was extraordinarily prevalent in both male and female Pima Indians. In males the disease frequency increased with age, with 66.7 per cent of those aged 65 years and over affected; in females the prevalence of disease was strikingly high after the age of 24 and reached a remarkable 92.6 per cent in those 65 and over.

As Table 3 indicates, 6 of the 21 Pima males with disease had previously been diagnosed by cholecystectomy or an abnormal hospital gallbladder radiograph. Thus, 15 (30.0 per cent) of 50 Pima males without known gallbladder disease had abnormal cholecystograms. Of 92 affected females, 54 were previously known; thus, abnormal radiographs were demonstrated in 38 (46.3 per cent) of the 82 Pima females without previously diagnosed disease.

TABLE 2. Prevalence of gallbladder disease in Pima Indians

AGE	NO. EXAMINED	WITH GALLBLADDER DISEASE NO.	%
Males			
15–24	5	0	0.0
25–34	6	0	0.0
35–44	9	1	11.1
45–54	10	4	40.0
55–64	11	6	54.5
65 and over	15	10	66.7
Total	56	21	37.5
Females			
15–24	11	1	9.1
25–34	18	13	72.2
35–44	33	22	66.7
45–54	28	19	67.9
55–64	19	12	63.2
65 and over	27	25	92.6
Total	136	92	67.6

TABLE 3. Diagnosis of gallbladder disease in sample

METHOD OF DOCUMENTATION	MALES	FEMALES	TOTAL
Total examined to date	56	136	192
Normal cholecystogram at CFSU Clinic	35	44	79
Number with gallbladder disease	21	92	113
Cholecystectomy	5	41	46
Abnormal cholecystogram at hospital	1	13	14
Gallstones	0	2	2
Two nonvisualizations	0	7	7
One nonvisualization	1	4	5
Abnormal cholecystogram at CFSU Clinic	15	38	53
Gallstones	3	14	17
Two nonvisualizations	7	9	16
One nonvisualization	5	15	20

In 5 males and 15 females who were examined at the Clinical Field Studies Unit Clinic, the diagnosis of gallbladder disease was based on only a single nonvisualization. However, when cholecystograms were repeated on these 20 cases, 17 (85.0 per cent) had either second nonvisualizations or gallstones on the repeat radiographs.

Morbidity from gallbladder disease

In Table 4 the clinical importance of gallbladder disease among Pima Indians is shown. Among males, "silent" gallstones were relatively frequent: only 6 (28.6 per cent) of the 21 affected males related "gallbladder symptoms"[4] or had a history of such symptoms noted in their medical records. Among the females, however, gallbladder disease resulted in considerable morbidity: 39 (42.4 per cent) of the 92 affected females had had a previous cholecystectomy and an additional 25 (27.2 per cent) had had gall-

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[4] Defined as an affirmative answer to the question "Have you ever had pain in your stomach or vomited after eating certain kinds of food?"

TABLE 4. Morbidity from gallbladder disease in Pima Indians

	MALES		FEMALES		TOTAL	
	NO.	%	NO.	%	NO.	%
Total examined	56	100.0	136	100.0	192	100.0
With gallbladder disease	21	37.5	92	67.6	113	58.9
Postprandial abdominal pain or nausea	6	10.7	64	47.1	70	36.5
Cholecystectomy	5	8.9	39	28.7	44	22.9
Abn. cholecystogram (hospital)	1	1.8	13	9.6	14	7.3
Abn. cholecystogram (CFSU)	0	0.0	12	8.8	12	6.3
No postprandial distress	15	26.8	28	20.6	43	22.4
With no gallbladder disease	35	62.5	44	32.4	79	41.1
Postprandial abdominal pain or nausea	5	8.9	7	5.1	12	6.3
No postprandial distress	29	51.8	33	24.3	62	32.3
No information	1	1.8	4	2.9	5	2.6

bladder symptoms but still retained their gallbladders. Thus, 64 (69.6 per cent) of the 92 affected females in this sample were having or had had clinical manifestations of gallbladder pathology at the time of the examination. The actual magnitude of gallbladder disease as a clinical problem among Pima women is best illustrated by the fact that 64 (47.1 per cent) of the 136 females in this sample had symptomatic gallbladder disease.

Discussion

These data, though as yet incomplete, indicate that the gallbladder was an extremely frequent site of pathology among Pima Indian females. Gallbladder disease was responsible for numerous hospitalizations, which often culminated in a major surgical procedure with its attendant morbidity and slight mortality. Unfortunately, we have no data on complications that may have compounded the morbidity from gallbladder disease in the members of this sample.

Gallbladder disease was less common among Pima males than among the females. Fewer males admitted to symptoms and fewer had had surgery. The earlier onset of gallbladder disease and the more frequent occurrence of biliary symptoms in females may perhaps be due to the effects of female hormones (*10*) on gallbladder physiology.

An analysis of other factors possibly contributing to gallbladder disease in the Pima—such as diet, pregnancy, obesity, diabetes, and serum cholesterol—will be reported when a higher proportion of this sample has been examined.

A review of the medical records of 575 members of the sample for evidence of documented gallbladder disease and possible associated conditions has previously been reported (*5*). In that study gallbladder disease was found to be related to increased parity but not to diabetes, body weight, or serum cholesterol except that in older Pima males there was an association with diabetes. These data must be interpreted cautiously, since the present study indicates that nearly a third of the Pima males and nearly half of the Pima females without documented clinical gallbladder disease actually had gallbladder pathology.

In order to advance the study of cholelithiasis and to further explore the problem of gallstones in Indians, it would be desirable to supplement the epidemiologic studies currently being performed among the Pima with selected biochemical and physiological investigations.

Fundamental to the concept of gallstone formation is the chemistry of the hepatic and gallbladder bile at the time stones are first being formed. In mice it has been possible to alter bile composition and to produce gallstones by feeding "lithogenic diets" (*3*). The biochemistry of human hepatic bile and the significance of the changes found in pathologic bile have recently been reviewed (*1*). Since other groups of American Indians appear to have a high prevalence of the disease, it is possible that the bile of American Indians, perhaps as a result of genetically determined factors, differs fundamentally in its composition from that found in other racial groups. To investigate this possibility, specimens of bile from Indians both with and without gallbladder disease should be analyzed. These could be obtained from the gallbladder at the time of laparotomy for some unrelated condition and at the time of cholecystectomy. Bile obtained at autopsy would be suitable for study only if it could be determined that its composition in the post mortem state was similar to bile examined before death, and particularly if the information could be supplemented with pertinent ante mortem data. An analysis of the constituents of gallbladder bile and their relation to age and sex in this gallstone-prone population, together with a comparison of bile from Indians with that of other racial groups, might add significantly to knowledge concerning the biochemical factors predisposing to cholelithiasis.

The Pima Indians have low levels of serum cholesterol (*5*), even though their diet seems to be high in saturated fats (*8*). It is possible that an unusually efficient mechanism for clearing serum cholesterol may be present, which might be mediated through exaggerated biliary excretion of cholesterol. Studies of the intake and absorption of dietary fats and of the synthesis and excretion of cholesterol might give clues to the etiology of gallbladder disease in this group.

It has been reported that peptic ulcers are rare among southwestern Indians (*9*). This observation may be relevant to the excess of gallstones among Indians, since a recent study found gastric secretion to be diminished in the majority of patients with cholelithiasis (*4*). Further studies relating gastric secretion to gallbladder function in Pima Indians appear to be indicated.

Summary

A preliminary report of a survey based on oral cholecystograms on a population of Pima Indians from Arizona has been made. The prevalence of gallbladder disease among those examined to date was significantly higher than had previously been reported, with 21 (37.5 per cent) of 56 males and 92 (67.6 per cent) of 136 females affected. Clinical symptoms attributable to gallbladder disease were found among 10.7 per cent of the 56 males and 47.1 per cent of the 136 females. Nearly a third of the males and nearly half of the females without previously known disease had abnormal cholecystograms.

Further biochemical and physiologic studies to supplement the current epidemiologic investigations are suggested.

Acknowledgments

We wish to thank the Tribal Council of the Gila River Indian Community and the Division of Indian Health of the United States Public Health Service for their encouragement and assistance. Special thanks are due to Drs. W. J. Held and Jon E. Gustafson, the radiologists at the Phoenix Indian Hospital who read the cholecystograms.

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HYPERGLYCEMIA IN PIMA INDIANS: A PRELIMINARY APPRAISAL OF ITS SIGNIFICANCE

Max Miller, Peter H. Bennett, and Thomas A. Burch[1]

There have been several reports in recent years indicating that certain groups of North American Indians have a high prevalence of diabetes mellitus (*5, 14*). It has also been suggested that the frequency and types of the associated clinical manifestations and complications differ in the American Indian from those observed in the Caucasian and the Negro (*12*). This report will examine the frequency of hyperglycemia and related variables in a North American Indian tribe, the Pima Indians of Arizona.

Methods

The Pima Indians have resided for at least several centuries in the region of the Gila River in south-central Arizona, which is typical of the hot, dry Sonoran desert of the southwestern United States. The Pima were traditionally an agricultural group, and they or their forebears, with the aid of irrigation water from the Gila River, have farmed its valley for about two thousand years (*4*). The majority of them now reside on the Gila River Indian Reservation, to the south and east of Phoenix, Arizona.

The inhabitants of the reservation reside in two main regions, one of which receives its medical care from the U.S. Public Health Service Indian Hospital in Phoenix and the other from the PHS Indian Hospital in Sacaton, Arizona. The latter region contains several villages that form an independent natural population group, geographically isolated from the other. Some 3,300 Indians aged 5 years and over reside in this community; of these 3,041 claim to be half- to full-blooded Pima.

Between 1 March 1965 and 1967, 1881 of these Pima Indians received a modified glucose tolerance test at the National Institutes of Health Clinic at the Sacaton PHS Indian Hospital. An oral 75-gram glucose equivalent carbohydrate load[2] (*6*) was given to each subject who attended the examination center regardless of the time of the previous meal. Two hours later venous blood was drawn into a tube containing sodium fluoride and another specimen was placed in a plain glass tube. Glucose concentration was measured on the AutoAnalyzer, using the modified Hoffman method,[3] in plasma obtained from the fluoride tube. Serum creatinine was determined on the additional serum sample.

Before drinking the carbohydrate load each subject was asked to void, and two hours later a urine sample was collected, which was tested for protein and glucose with dipsticks.[4] Whenever protein was found in a concentration of approximately 30mg/100ml or more ($\geq 1+$), a quantitative determination of the urinary protein concentration in a sample of the urine was made together with determination of the urinary creatinine concentration. Among those aged 15 years and over a subsample was examined more thoroughly and retinal photographs, electrocardiograms, and radiographs of the soft tissues of the left calf (lateral view) and thigh (AP view) were taken.[5]

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[1] Presented by Dr. Miller.

[2] Glucola, Ames Company, Elkhart, Indiana, or Dexcola, Custom Laboratories, Baltimore, Maryland.

[3] Technicon AutoAnalyzer Method File N-20, Technicon Instruments Corporation, Chauncey, New York, 1965.

[4] Labstix, Ames Company, Elkhart, Indiana.

A prospective study of the outcome of the pregnancies among the Pima women living in the same community was initiated in October 1965. The modified glucose tolerance test was performed on these women one to three days after parturition, and data on the course of pregnancy and the characteristics of the offspring were collected. In addition, a retrospective study was made of all previous pregnancies and their outcome among the Pima women aged 25 to 44 years on 1 January 1966 (*1*).

Autopsy data were collected on members of the Pima Indian population who have died since our initial survey in 1963, and death certificates have been collected systematically since 1964.

Results

Table 1 shows the number of Pima subjects in the sample with hyperglycemia by age and sex and the number with previously diagnosed diabetes who have received specific hypoglycemic therapy in the course of routine medical care. The prevalence of previously treated diabetes was highest in the males aged 65 to 74 (26.2 per cent) and in the 55-to-64-year-old females (40.8 per cent).

If "diabetes" is arbitrarily defined as a venous plasma glucose level of 160mg/100ml or more two hours after a 75-gram glucose equivalent load, the majority of cases of the disease were

TABLE 1. Carbohydrate intolerance among Pima (4/8–8/8) Indians of the Gila River Reservation aged 5 years and over, 1965–1967

AGE GROUP BY SEX	NO. EXAMINED	NO. WITH KNOWN PREVIOUSLY TREATED DIABETES	NO. WITH TWO-HOUR GLUCOSE LEVELS ≥ 160 MG	NO. WITH "DIABETES"	% WITH "DIABETES"
Males					
5–14	238	0	0	0	0.0
15–24	146	0	4	4	2.7
25–34	86	0	15	15	17.4
35–44	91	10	20	30	33.0
45–54	70	13	17	30	42.9
55–64	74	16	15	31	41.9
65–74	61	16	12	28	45.9
75 and over	38	4	12	16	42.1
TOTAL	804	59	95	154	19.2
Females					
5–14	284	0	7	7	2.5
15–24	230	1	11	12	5.2
25–34	149	9	21	30	20.1
35–44	150	23	50	73	48.7
45–54	93	25	34	59	63.4
55–64	93	38	26	64	68.8
65–74	55	16	15	31	56.4
75 and over	23	3	9	12	52.2
TOTAL	1077	115	173	288	26.7

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[5] The subsample consisted initially of a one-in-two sample of those aged 30 years and over but was extended, after examination of the first 1,100 persons, to include all those examined aged 15 years and over. Though this is not a truly random sample, we believe that it is unlikely to be significantly biased in relation to any findings associated with plasma glucose levels.

undiagnosed at the time of the modified glucose tolerance test. The combined prevalence of previously and newly diagnosed "diabetes" reached a maximum of 45.9 per cent in the males aged 65 to 74 years and 68.8 per cent in the females aged 55 to 64. In all age groups, especially those of 35 years and over, the prevalence of "diabetes" in the females exceeded the rate in the males, the differences being significant in each decade between 35 and 64 years. Thus, both male and female Pima Indians had an extraordinary frequency of "diabetes"—higher than that reported in any other population studied. Table 2 and Figure 1 show the distribution of the two-hour plasma glucose results on all Pima Indians examined regardless of whether they had previously diagnosed diabetes, provided they did not have concurrently treated diabetes—i.e., had not taken specific hypoglycemic medication within the period in which the two-hour glucose determination could have been directly influenced.[6] Such a determination was made on 1,776 of the subjects. The age- and sex-specific cumulative frequency distributions of the plasma glucose values indicated that there was a relatively steep rise in the cumulative frequency distribution in the lower range of the glucose values in all age groups. There was not, however, a uniform increase in each successive age group at all percentile levels. Below the median (fiftieth percentile level) in both sexes the increases were relatively small, but the upper percentile glucose values were much higher in the older age groups of both sexes than in the younger subjects. This suggests that, as age increased, the proportion of subjects with hyperglycemia increased markedly.

From this the inference was drawn that the plasma glucose levels in all except the youngest groups of each sex were not homogeneous but consisted of two or more components. Further analysis indicated that in each sex and in all decades from 25 to 74 years the frequency distribution of the logarithms of the two-hour glucose values had a bimodal distribution. Using an iterative maximum likelihood procedure, these distributions were dissected mathematically and each of the observed distributions in those aged 25 to 74 years—for example, that shown in Figure 2 for males aged 55 to 74—proved to be a statistically satisfactory fit for two overlapping $\log_{10}$ normal (Gaussian) distribution curves. In those below 25 years, however, only a unimodal distribution was found, as is shown in Figure 3 for males aged 15 to 24, which was a satisfactory fit for a single $\log_{10}$ normal distribution (*13*). Hence the heterogeneity inferred from the cumulative percentage distribution in the older groups appeared to result from the presence of

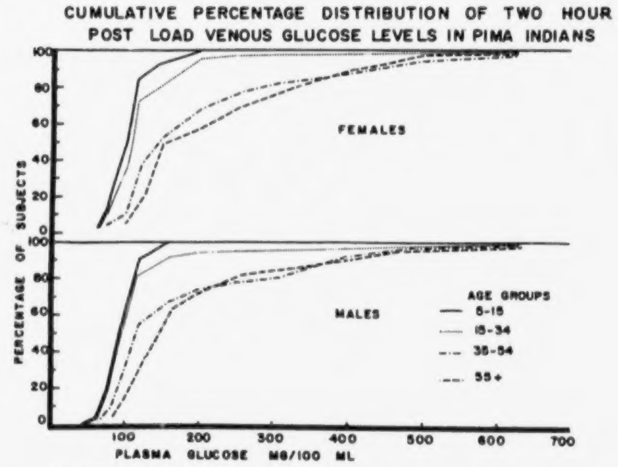

FIGURE 1. Age- and sex-specific cumulative percentage distributions of two-hour post-load venous plasma glucose levels in Pima Indians

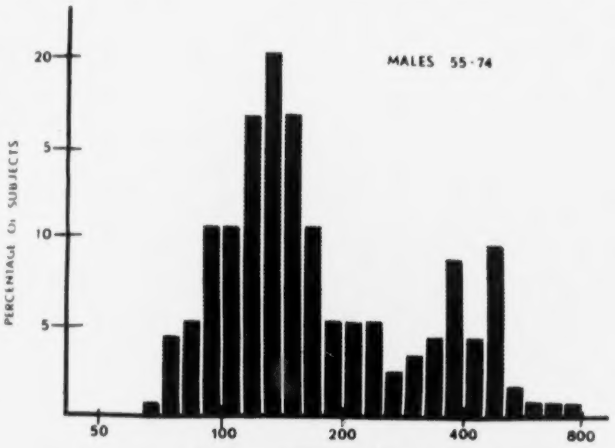

FIGURE 2. Frequency distribution of two-hour post-load venous plasma glucose levels (logarithmic scale) in Pima Indian males aged 55–74 years

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[6] For patients taking tolbutamide or phenformin this period was 24 hours; for patients taking insulin or chlorpropamide or other oral agents or combinations of oral agents, 48 hours.

TABLE 2. Cumulative percentage of two-hour post-load venous plasma glucose levels (mg/100ml) in Pima Indians

PLASMA GLUCOSE LEVEL	MALES								FEMALES							
	5–14 YEARS		15–34 YEARS		35–54 YEARS		55 AND OVER		5–14 YEARS		15–34 YEARS		35–54 YEARS		55 AND OVER	
	NO.	CUM. %	NO.	CUM. %	NO.	CUM. %	NO.	CUM. %	NO.	CUM. %	NO.	CUM. %	NO.	CUM. %	NO.	CUM. %
0–50	1	0.4	2	0.9	1	0.7	—	—	—	—	—	—	—	—	—	—
–63	6	2.9	6	3.4	2	2.0	—	—	5	1.8	8	2.1	—	—	1	0.8
–80	36	18.1	33	17.7	11	9.3	6	3.9	31	12.7	29	9.9	9	4.2	2	2.3
–100	86	54.2	76	50.4	26	26.5	18	15.5	98	47.2	94	35.1	12	9.9	4	5.4
–126	87	90.8	70	80.6	43	55.0	34	37.4	106	84.5	138	72.1	59	37.6	19	20.0
–159	22	100.0	26	91.8	18	66.9	40	63.2	37	97.5	68	90.3	31	52.1	38	49.2
–199	—	—	5	94.0	11	74.2	18	74.8	7	100.0	18	95.2	36	69.0	11	57.7
–251	—	—	3	95.3	6	78.1	11	81.9	—	—	9	97.6	17	77.0	15	69.2
–316	—	—	2	96.1	4	80.8	5	85.2	—	—	—	—	12	82.6	11	77.7
–398	—	—	3	97.4	18	92.7	9	91.0	—	—	1	97.9	11	87.8	16	90.0
–501	—	—	3	98.7	6	96.7	11	98.1	—	—	5	99.2	12	93.4	6	94.6
–631	—	—	2	99.6	4	99.3	1	98.7	—	—	3	100.0	11	98.6	4	97.7
–796	—	—	1	100.0	—	—	2	100.0	—	—	—	—	1	99.1	3	100.0
–1000	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
–1260	—	—	—	—	1	100.0	—	—	—	—	—	—	2	100.0	—	—
	238		232		151		155		284		373		213		130	
With concurrently treated diabetes	0		0		10		18		—		6		30		41	
Total	238		232		161		173		284		379		243		171	

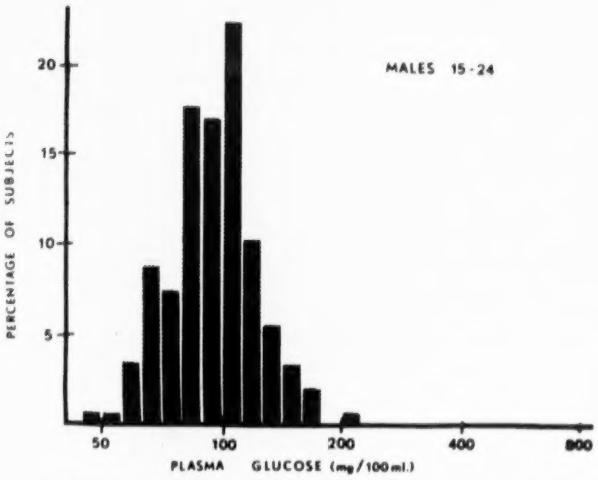

FIGURE 3. Frequency distribution of two-hour post-load venous plasma glucose levels (logarithmic scale) in Pima Indian males aged 15–24 years

two components, one characterized by a much higher mean plasma glucose level than the other.

The parameters of the distributions were such that bimodality was apparent only because the proportion of subjects who contributed to the higher curve was relatively high.

In order to determine whether the subjects in the two distributions consisted of those with and without diabetes mellitus in the clinical sense, the relationship of other parameters of this disease to glucose tolerance levels was examined.

Diabetic retinopathy

"Diabetic retinopathy" is allegedly one of the most specific signs of diabetes mellitus. Its frequency was assessed in the Pima Indians by the evaluation of retinal photographs for the presence of microaneurisms, preretinal and vitreous hemorrhages, exudates, and neovascularization. Table 3 shows the frequency of diabetic retinopathy in all subjects aged 15 years and over who had one or more satisfactory retinal photographs.

No definite evidence of diabetic retinopathy was observed in male or female Pima Indians under 35. In those above this age, it was found only in those with the higher plasma glucose levels. In those with two-hour plasma glucose levels of 200mg/100ml and over, retinopathy was approximately twice as frequent as in those with levels between 160 and 199mg/100ml. Among those with diabetes concurrently under treatment, retinopathy was observed in 20.0 per cent of the males and in 19.6 per cent of the females.

Proteinuria

Other evidence that hyperglycemia among the Pima Indians was associated with vascular complications was investigated by examination for evidence of renal disease. Table 4 shows the relationship of proteinuria ($\geq 1+$) to the two-hour post-load plasma glucose level. The frequency of proteinuria among both males and females increased with age and plasma glucose level. Within each age group there was a gradient of increasing frequency of proteinuria with increasing severity of hyperglycemia. This relationship was similar in males and females, as was the over-all frequency of proteinuria in both sexes and in every age group of each sex.

In 105 of the 128 subjects with proteinuria it was possible to make more quantitative estimates of proteinuria by determining albumin and creatinine concentrations in the two-hour urine samples. Since the daily excretion of creatinine in the urine is relatively constant (*11*), the albumin/creatinine (A/C) ratio will yield an approximate estimate of the 24-hour urinary excretion of protein. Thus, an A/C ratio of 1 or more indicates that one or more grams of protein are excreted daily.

Markedly elevated A/C ratios (≥ 1.0) were found more frequently in those who had two-hour post-load glucose levels in excess of 200mg/100ml and in the concurrently treated diabetics than in subjects with lower values (Table 5). Thus, among those with high glucose values, proteinuria was both more frequent in any degree and also much more severe.

The nephrotic syndrome associated with severe intercapillary glomerulosclerosis is recognized as one of the important complications of the renal disease of diabetes mellitus. The essential feature of this syndrome is the occurrence of excessive proteinuria, usually exceeding 3 grams a day. It is noteworthy that of those with plasma glucose values of 200mg/100ml and over, 11 of the 39 with proteinuria on whom the

TABLE 3. Diabetic retinopathy and two-hour post-load plasma glucose levels in Pima (4/8–8/8) Indians examined 1965–1967

AGE GROUP BY SEX	PLASMA GLUCOSE LEVEL (MG/100ML) 0–100	101–159	160–199	200+	WITH CONCURRENTLY TREATED DIABETES	TOTAL
Males						
15–34 years						
No. examined	43	43	0	8	1	95
No. affected	0	0	0	0	0	0
Per cent	0.0	0.0	0.0	0.0	0.0	0.0
35–54 years						
No. examined	26	35	9	27	9	106
No. affected	0	0	0	4	2	6
Per cent	0.0	0.0	0.0	14.8	22.2	5.7
55 and over						
No. examined	15	41	11	19	10	96
No. affected	0	0	1	3	2[a]	6
Per cent	0.0	0.0	9.1	15.8	20.0	6.3
Total						
No. examined	84	119	20	54	20	297
No. affected	0	0	1	7	4	12
Per cent	0.0	0.0	5.0	12.9	20.0	4.0
Females						
15–34 years						
No. examined	43	71	6	12	3	135
No. affected	0	0	0	0	0	0
Per cent	0.0	0.0	0.0	0.0	0.0	0.0
35–54						
No. examined	13	67	27	45	23	175
No. affected	0	0	0	4	3	7
Per cent	0.0	0.0	0.0	8.9	13.0	4.0
55 and over						
No. examined	2	29	6	30	20	87
No. affected	0	0	1	6	6	13
Per cent	0.0	0.0	16.7	20.0	30.0	14.9
Total						
No. examined	58	167	39	87	46	397
No. affected	0	0	1	10	9	20
Per cent	0.0	0.0	2.6	11.5	19.6	5.3

[a] Includes one male who at the time of the retinal photographs had a plasma glucose of 156 mg/100 ml while not on medication. He had, however, lost weight and had carcinoma stomach with metastases, and in 1965 had a fasting blood glucose of 165 mg/100 ml.

TABLE 4. Proteinuria and two-hour post-load plasma glucose levels in Pima (4/8–8/8) Indians examined 1965–1967 (≥1+ on dipsticks [4])

AGE GROUP BY SEX	PLASMA GLUCOSE LEVEL (MG/100ML)				WITH CONCURRENTLY TREATED DIABETES	TOTAL
	0–100	101–159	160–199	200+		
Males						
15–34 years						
No. examined	108	90	5	13	0	216
No. affected	6	5	0	1	0	12
Per cent	5.6	5.6	0.0	7.7	0.0	5.6
35–54 years						
No. examined	39	59	10	36	10	154
No. affected	2	6	2	4	4	18
Per cent	5.1	10.2	20.0	11.1	40.0	11.7
55 and over						
No. examined	22	70	17	35	17	161
No. affected	2	6	3	10	6	27
Per cent	9.1	8.6	17.6	28.6	35.3	16.8
Total						
No. examined	169	219	32	84	27	531
No. affected	10	17	5	15	10	57
Per cent	5.9	7.8	15.6	17.9	37.0	10.7
Females						
15–34 years						
No. examined	109	176	16	17	6	324
No. affected	4	7	2	5	0	18
Per cent	3.7	4.0	12.5	29.4	0.0	5.6
35–54 years						
No. examined	20	86	36	64	29	235
No. affected	0	4	2	10	5	21
Per cent	0.0	4.7	5.6	15.7	17.2	8.9
55 and over						
No. examined	8	51	10	44	40	153
No. affected	2	2	0	15	13	32
Per cent	25.0	3.9	0.0	31.8	32.5	20.3
Total						
No. examined	137	313	62	125	75	712
No. affected	6	13	4	30	18	71
Per cent	4.4	4.2	6.5	24.0	24.0	10.0

TABLE 5. Albumin/creatinine ratios in subjects with $\geq 1+$ proteinuria

	PLASMA GLUCOSE LEVEL (MG/100ML)				WITH CONCURRENTLY TREATED DIABETES	TOTAL
	0–100	101–159	160–199	200+		
Males						
No. examined	5	17	4	13	9	48
$A/C \geq 1.0$	0	2	2	9	7	20
Per cent	0.0	11.7	50.0	69.2	77.8	41.7
Females						
No. examined	5	10	2	26	14	57
$A/C \geq 1.0$	2	2	0	14	11	29
Per cent	40.0	20.0	0.0	53.8	78.6	50.9
Total						
No. examined	10	27	6	39	23	105
$A/C \geq 1.0$	2	4	2	23	18	49
Per cent	20.0	14.8	33.3	58.9	78.3	46.7

A/C ratios were determined had values of 3.0 and over, as did 11 of the 23 with concurrently treated diabetes. A/C ratios of this magnitude were not encountered in subjects with two-hour glucose levels of less than 200mg/100ml except in one 32-year-old male who was known to have had acute glomulonephritis nine years previously.

Serum creatinine levels

In order to determine the frequency of subjects with significant reductions in renal function, serum creatinine levels were examined. Elevated levels were defined for this purpose as 1.5mg/100ml and over in males, and 1.3mg/100ml and over in females. Such levels were found mainly among subjects with two-hour plasma glucose levels of 200mg/100ml and over and among those with concurrently treated diabetes (Table 6). They occurred predominantly among the older subjects in the same groups in which marked proteinuria was found.

The records of four male subjects with plasma glucose levels of 101 to 200mg/100ml who had elevated serum creatinine levels were examined to determine possible reasons for these. Two had histories of hypertension and congestive heart failure, and another had received renal dialysis following trauma and shock. The fourth, with a glucose value of 160–199mg/100ml, was a known diabetic of many years' duration. He had previously had several blood glucose values in excess of 250mg/100ml and had received insulin and tolbutamide for many years. Prior to testing he had lost at least 26 pounds in weight, and he died 23 days after examination.

Thus, evidence was found that hyperglycemia among the Pima Indians was strongly associated with proteinuria and impairment of renal function, especially among the older subjects of both sexes.

Vascular calcification

Evidence of disease of the peripheral vasculature was evaluated in soft-tissue radiographs of the thigh (femoral artery) and the calf (tibial arteries), since vascular calcification has been shown to be more frequent among diabetics than among nondiabetics (*3*) and to develop concomitantly with increasing duration of diabetes among the younger subjects with diabetes (*15*). Tables 7 and 8 show the frequency of calcification in these vessels. In both sites calcification was found more frequently in the males than in the females, was age-related, and was encountered more frequently in the tibial arteries than in the femoral artery. The calcification observed was mainly medial in type; the intimal type was rarely seen and never occurred alone.

Calcification in the femoral artery and in the tibial arteries was not found in any Pima Indians below the age of 35 years. In the older groups

TABLE 6. Elevated serum creatinine and two-hour post-load plasma glucose levels in Pima (4/8–8/8) Indians examined 1965–1967 (Males ≥1.5 mg/100 ml : Females≥1.3 mg/100 ml)

AGE GROUP BY SEX	PLASMA GLUCOSE LEVEL (MG/100ML)				WITH CONCURRENTLY TREATED DIABETES	TOTAL
	0–100	101–159	160–199	200+		
Males						
15–34 years						
No. examined	84	61	2	8	0	155
No. affected	0	0	0	0	0	0
Per cent	0.0	0.0	0.0	0.0	0.0	0.0
35–54 years						
No. examined	27	49	9	30	10	125
No. affected	0	2[a]	0	0	2	4
Per cent	0.0	4.1	0.0	0.0	20.0	3.2
55 and over						
No. examined	19	64	17	26	16	142
No. affected	0	1[a]	1[a]	2	1	5
Per cent	0.0	1.6	5.9	7.7	6.3	3.5
Total						
No. examined	130	174	28	64	26	422
No. affected	0	3	1	2	3	9
Per cent	0.0	1.7	3.6	3.1	11.5	2.1
Females						
15–34 years						
No. examined	62	84	6	11	2	165
No. affected	0	0	0	1	0	1
Per cent	0.0	0.0	0.0	9.1	0.0	0.6
35–54 years						
No. examined	14	67	26	52	26	185
No. affected	0	0	0	0	2	2
Per cent	0.0	0.0	0.0	0.0	7.7	1.1
55 and over						
No. examined	6	48	6	41	34	135
No. affected	0	0	0	5	4	9
Per cent	0.0	0.0	0.0	12.2	11.8	6.7
Total						
No. examined	82	199	38	104	62	485
No. affected	0	0	0	6	6	12
Per cent	0.0	0.0	0.0	5.8	9.7	2.5

[a] See text.

TABLE 7. Medial calcification of the femoral artery and two-hour post-load plasma glucose levels in Pima (4/8–8/8) Indians examined 1965–1967

AGE GROUP BY SEX	PLASMA GLUCOSE LEVEL (MG/100ML) 0–100	101–159	160–199	200+	WITH CONCURRENTLY TREATED DIABETES	TOTAL
Males						
15–34 years						
No. examined	59	49	3	7	0	118
No. affected	0	0	0	0	0	0
Per cent	0.0	0.0	0.0	0.0	0.0	0.0
35–54 years						
No. examined	23	38	5	28	9	103
No. affected	0	0	1	2	1	4
Per cent	0.0	0.0	20.0	7.1	11.1	3.9
55 and over						
No. examined	17	59	14	26	15	131
No. affected	5	17	5	7	8	42
Per cent	29.4	28.8	35.7	26.9	53.3	32.1
Total						
No. examined	99	146	22	61	24	352
No. affected	5	17	6	9	9	46
Per cent	5.1	11.6	27.3	14.8	37.5	13.1
Females						
15–34 years						
No. examined	39	68	10	11	3	131
No. affected	0	0	0	0	0	0
Per cent	0.0	0.0	0.0	0.0	0.0	0.0
35–54 years						
No. examined	12	54	24	49	22	161
No. affected	0	0	0	2	0	2
Per cent	0.0	0.0	0.0	4.1	0.0	1.2
55 and over						
No. examined	5	42	7	36	34	124
No. affected	0	2	0	7	6	15
Per cent	0.0	4.8	0.0	19.4	17.6	12.1
Total						
No. examined	56	164	41	96	59	416
No. affected	0	2	0	9	6	17
Per cent	0.0	1.2	0.0	9.4	10.2	4.1

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TABLE 8. Medial calcification in the tibial arteries and two-hour post-load plasma glucose levels in Pima (4/8–8/8) Indians examined 1965–1967

AGE GROUP BY SEX	PLASMA GLUCOSE LEVEL (MG/100ML)				WITH CONCURRENTLY TREATED DIABETES	TOTAL
	0–100	101–159	160–199	200+		
Males						
15–34 years						
No. examined	58	47	3	7	0	115
No. affected	0	0	0	0	0	0
Per cent	0.0	0.0	0.0	0.0	0.0	0.0
35–54 years						
No. examined	21	38	5	28	9	101
No. affected	0	2	1	6	2	11
Per cent	0.0	5.3	20.0	21.4	22.2	10.9
55 and over						
No. examined	16	56	14	26	14	126
No. affected	4	24	5	11	8	52
Per cent	25.0	42.9	35.7	42.3	57.1	41.3
Total						
No. examined	95	141	22	61	23	342
No. affected	4	26	6	17	10	63
Per cent	4.2	18.4	27.3	27.9	43.5	18.4
Females						
15–34 years						
No. examined	45	68	10	11	3	137
No. affected	0	0	0	0	0	0
Per cent	0.0	0.0	0.0	0.0	0.0	0.0
35–54 years						
No. examined	12	58	21	49	23	163
No. affected	0	0	0	2	0	2
Per cent	0.0	0.0	0.0	4.1	0.0	1.2
55 and over						
No. examined	5	42	7	34	32	120
No. affected	0	3	2	10	7	22
Per cent	0.0	7.1	28.6	29.4	21.9	18.3
Total						
No. examined	62	168	38	94	58	420
No. affected	0	3	2	12	7	24
Per cent	0.0	1.8	5.3	12.8	12.1	5.7

both sites were affected more frequently, but not exclusively, in subjects with the higher two-hour plasma glucose values and with concurrently treated diabetes. In the majority of subjects with femoral artery involvement, the tibial arteries were also affected.

Hyperglycemia and pregnancy

Diabetes mellitus is well recognized as having characteristic effects on pregnancy and its outcome. In order to determine whether similar effects were found in the Pima Indians a review was made of the past pregnancies of females aged 25 to 44 years on 1 January 1966 who had received a modified glucose tolerance test and had borne at least one child. On the basis of this recent test and clinical history, the females were divided into three groups: those with two-hour post-load venous plasma glucose levels of less than 140mg/100ml, those with either clinical diabetes requiring hypoglycemic treatment or plasma glucose values of 160mg/100ml or over, and those who had intermediate plasma glucose levels. The last group were considered to be in a "gray zone" and were not analyzed. Pregnancies among the second group were divided into those that occurred before the clinical diagnosis of diabetes ("prediabetic") and those that occurred after the diagnosis ("diabetic"). The results appear in Table 9. Among the Pima females studied, 47 (3.8 per cent) of the 1,253 pregnancies were considered "diabetic," as against a frequency in the Cleveland, Ohio, metropolitan area of 0.28 per cent (*8*). The frequency of "diabetic" pregnancy was therefore some 10 to 15 times greater among the Pima Indians than among the women of Cleveland.

The frequency of offspring weighing nine pounds or more at birth was significantly higher in the Pima "diabetic" group than in the "normoglycemic" mothers. The frequency of congenital anomalies and the perinatal mortality rate were also significantly higher among the "diabetic" group. The perinatal mortality rate in the latter was 25.5 per cent, which is in the same range as that reported in other populations (*10*).

A prospective study of the current pregnancies of Pima women was started in 1965. The modified glucose tolerance test was performed on the mother generally two to three days postpartum, but in a few instances it was done on the first day. Table 10 shows the relationship of the infant birth weight to the two-hour post-load plasma venous glucose level among such women. While the number with high postpartum glucose values was small, there was a highly significant relationship between birth weight and maternal postpartum glucose level, an association consistent with that noted in the retrospective study.

TABLE 9. Outcome of pregnancy among 215 Pima females (retrospective study)

OUTCOME	"NORMOGLYCEMIC"	"PREDIABETIC"	"DIABETIC"	TOTAL
Birth weight				
No. of offspring	571	229	38	838
≥9 pounds at birth	37	26	15	78
Per cent	6.5	11.4	42.9	9.3
Congenital anomalies				
No. of offspring	785	375	47	1207
With congenital anomalies	31	13	9	53
Per cent	3.9	3.5	19.1	4.4
Perinatal mortality				
No. of births	815	391	47	1253
No. of deaths [a]	10	12	12	34
Per cent	1.2	3.1	25.5	2.7

[a] Includes stillbirths and neonatal deaths occurring in first 28 days of life.

TABLE 10. Infant birth weight and maternal post partum two-hour post-load plasma glucose levels in Pima (4/8–8/8) Indians examined Oct. 1965–Dec. 1967

MATERNAL PLASMA GLUCOSE (MG/100ML)	NO. OF PREGNANCIES	OFFSPRING WEIGHING ≥4000 GRAMS AT BIRTH NO.	%
0– 63	17	0	0.0
64– 80	49	2	4.1
81–100	90	5	5.6
101–126	76	5	6.6
127–159	33	9	27.3
160–199	6	2	33.3
200+	3	1	
Total	274	24	8.8

Mortality

Death certificates have been collected on the Pima Indians of the Gila River Indian Reservation since 1964, and autopsy data were obtained on persons who have died since 1963. About half of those who died received post mortem examinations. Over a four-year period there were 53 male and 49 female deaths among persons aged 15 years and over who had available glucose estimations or whose diabetic status was known, excluding those due to accidents or violence (Table 11).

For comparative purposes the subjects were classified as "diabetic" on the basis of a clinical history of diabetes and/or high glucose values. Age-standardized death rates for subjects with "diabetes" in both sexes were found to be higher than those of the "nondiabetics," although the diagnosis was sometimes based upon less-than-ideal criteria. The rate was approximately twice as high in the females and at least 40 per cent higher in the males with "diabetes." The findings indicate that mortality rates among Pima subjects with hyperglycemia were substantially higher than among those with "normal" plasma glucose levels.

TABLE 11. Four-year mortality, excluding accidental and violent death (1964–1967) in Pima Indians aged 15 years and over

	MALES	FEMALES
Total no. deaths	53	49
No. with diabetes	27	34
Age-standardized rates/ 100 persons		
Diabetics	11.2	9.8
Nondiabetics	7.8	4.4

Post-mortem study

Of the Pima Indians who died since our initial study in 1963, autopsy data were available on 34 subjects with "diabetes" and on 19 without. Evidence of nodular intercapillary glomulerosclerosis was reported in six of the "diabetics" and in none of the others. Seven of the 34 diabetics had myocardial infarcts, compared to 2 of the 19 "nondiabetics." Tuberculosis was found in 7 "diabetics" and in only 1 "nondiabetic." Pyelonephritis, howover, was reported as frequently among the "nondiabetics" as the "diabetics." Amputations had been performed on 2, and mucormycosis was present in 1 of those with "diabetes."

Discussion

The data presented show that hyperglycemia is extremely frequent among the Pima Indians. As *complete* glucose tolerance tests were not performed routinely and as only a single venous glucose determination made two hours after a 75-gram glucose equivalent load was available on all subjects, the usual criteria for the diagnosis of diabetes mellitus could not be applied.

In order that comparisons might be made with other studies, however, the frequency of subjects who had received specific treatment for diabetes was determined, as was the frequency of subjects who had two-hour venous *plasma* glucose levels of 160mg/100ml and over. It was felt that this level, which corresponds to a *whole-blood* sugar of approximately 140mg/100ml, was suitable for comparison with several studies in which somewhat similar testing methods were used. It was found that known diabetes and hyperglycemia, as so defined, occurred more frequently among the Pima Indians than in any other population thus far studied. Unusually high frequencies of diabetes have been found among the Cherokee Indians of North Carolina, but the prevalence observed in the Pima Indians was even greater (*14*). It was also very much greater than had been found among Alaskan Eskimos (*9*) and was 10 to 15 times greater than the rate believed to be representative of the United States as a whole (*7*).

To determine whether hyperglycemia in Pima Indians was associated with the chronic vascular complications that are characteristic of diabetes in other populations, analyses were made to discover the frequency of diabetic retinopathy, renal disease, and vascular calcification among Pima subjects aged 15 years and over as a function of their plasma glucose level or previously known diabetes. Retinopathy, as shown on fundus photographs, and impaired renal function, as indicated by elevated serum creatinine levels or heavy proteinuria (A/C ratio ≥ 3.0), were found exclusively among those with two-hour glucose levels in excess of 200mg/100ml or with concurrently treated diabetes, except in rare individuals in whom the findings were clearly explained on another basis. Proteinuria of all degrees was more frequent among those with high glucose levels or treated diabetes and, when present, was moderately severe (A/C ratio ≥ 1.0) four times as frequently as in those with glucose levels below 200mg/100ml.

Since retinopathy and nephropathy are considered highly specific findings in diabetes mellitus, their frequent occurrence in Pima Indians with elevated two-hour plasma glucose levels implies that the hyperglycemia in this population was a manifestation of the diabetic syndrome as recognized elsewhere. This conclusion was strengthened by the reports of cases of nodular intercapillary glomerulosclerosis at autopsy among such subjects.

On the other hand, disease of the larger vessels, as shown by the presence of vascular calcification in the lower limbs, occurred among older subjects over the whole range of glucose levels but was again more frequent among those of the same age and sex with glucose levels in the upper range or with concurrently treated diabetes. Standard twelve-lead electrocardiograms have also been performed on the same subjects, and the findings will be reported separately. Post mortem studies, however, have already indicated that myocardial infarction occurred twice as frequently among the "diabetics" in the autopsy series.

Over a four-year period the age-standardized mortality rate among Pima Indian subjects on whom glucose determination had been made, either in the course of routine medical care or for research purposes, was shown to be greater among those of each sex with the higher glucose levels than among those with lower levels. This finding was consistent with the increased mortality from diabetes mellitus reported in Joslin Clinic patients, which is mainly related to the chronic vascular complications (*2*). Though similar processes appear to be responsible for the excessive Pima mortality, the number of deaths is so far too small for deducing the relative importance of the various types of vascular complications. Serial studies of renal function and electrocardiograms as well as systematic autopsy, whenever possible, will make possible such a definition in the future.

The frequency of "diabetic" pregnancy among the Pima Indians was in accordance with that which might be predicted from the over-all prevalence of hyperglycemia. The demonstration of significantly increased perinatal mortality, high infant birth weight, and high frequency of congenital anomalies resulting from these pregnancies further implies that hyperglycemia in Pima Indians has the same pathologic significance as diabetes mellitus among Caucasians and Negroes (*8, 10*).

As the classic symptoms of diabetes mellitus are related mainly to the individual levels of hyperglycemia and are ameliorated by the specific hypoglycemic medications, the substantial proportion of subjects who had received such treatment may reflect the past frequency of the characteristic symptoms of diabetes mellitus among the Pima Indians. While only 40 per cent of the subjects with glucose values of 160mg/100ml and over had received such therapy, they included 60 per cent of the females and 50 per cent of the males aged 55 to 64 years with such levels. This observation suggests that substantial numbers of the younger persons with hyperglycemia will become symptomatic in the future.

Before the systematic study of the Pima Indian community was begun, diabetes mellitus was locally recognized as an important health problem but the true frequency of hyperglycemia and of the vascular manifestations were unknown. Although some other southwestern Indian tribes are recognized as having a high frequency of the disease, others are said to have a low prevalence. The significance of this variation, if it were confirmed and if the reasons for the differences could be identified, would be an important contribution to our understanding of the etiology of diabetes mellitus.

A definition of the geography of the disease in the southwestern American Indian seems to be needed as a matter of some urgency, since these emerging groups are likely to be subjected to radical and rapid change. Some may lose their identity; others may adopt ways widely different from those of their forebears. The study of differing tribes with high and low prevalences of diabetes at present could contribute to our knowledge of the etiology, especially if identical methods of evaluation could be used in several of the groups. Such investigations could be enhanced by parallel study of groups of the same race in Central and South America. As these groups are subjected to change and as their life expectation is increased, it is possible that others may go the same way: in them diabetes mellitus may emerge as a major chronic disease problem, as it is today among the Pima Indians.

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MALARIA IN THE AMERICAN INDIAN

George G. Giglioli

Malaria prevails, or has prevailed up to recently, over very wide areas of the Western Hemisphere, lying between latitudes 50° N and 40° S and from the shores of the Atlantic Ocean to those of the Pacific. The American Indian ranges from Canada to Patagonia, and some thirteen species of American anopheles mosquitoes have been linked with malaria transmission, seven species being vectors of major public health importance in one country or another. The range of ecological and epidemiological variation is therefore practically unlimited, and it would be quite impossible to submit a concise, comprehensive account of malaria in the American Indian, even if adequate information on the subject were available. Unfortunately, published data are conspicuously absent from the literature, and none of the principal malaria eradication services have been able to supply information on this special malariological problem.

During the great upsurge of antimalarial activities over the last twenty years, emphasis was placed on vector destruction and chemotherapy, not on epidemiology. Excessive hopes were founded on the new insecticides with long-lasting residual effect, and on the powerful antimalarial drugs developed during the war and in the early postwar period. Malaria eradication campaigns were launched widely and waged with little or no attempt to establish base-line data on the local epidemiology of the disease.

For obvious economical, political, and logistical reasons, efforts in the sparsely inhabited countries of the American tropics were concentrated on the more populous areas, no distinction being made in respect to the different ethnic groups that might be present. Given the magnitude of the task, the remote, scattered, and economically unimportant Indian communities were—to a great extent—missed, overlooked, or ignored.

The present discussion will be restricted to the Indians of Guyana, who are estimated to number 32,000, widely scattered over the least accessible parts of the country. At the present day, nine tribes are represented: the Warau of the coastal lowlands, mostly in the North-West District, adjoining the delta of the Orinoco; the Arawak, of the lowland forests of the near interior; the Wapishana (Arawak stock), of the South Rupununi Savannah; the Carib, formerly very numerous and widely distributed, but now reduced to very small numbers, mainly on the upper Barama and Barima rivers in the North-West District; the Akawai, of the upper Berbice, Demerara, Cuyuni, and Mazaruni rivers; the Patamona, of the southern portion of the Pakaraima Plateau; the Arekuna, of the upper Cuyuni and Mazaruni mountains and savannahs; the Makusi, of the North Rupununi Savannah; and the Wai-Wai, of the Essequibo headwaters. The last five tribes are of Carib stock.

Some of these tribes extend beyond the political frontiers into Venezuela and Brazil. Generalizations about malaria are not permissible, and my observations refer specifically to Guyana; it may be noted, however, that few environments present greater stability and uniformity than the equatorial rain forest, which covers most of the Guianas and Amazonia, where *Anopheles darlingi,* the most efficient malaria carrier in the Western Hemisphere, is the prevailing, and often the only, vector. It is in this vast region that the majority of American Indians live still fully exposed to the ravages of uncontrolled and untreated malaria.

The study of racial differences in respect to the epidemiology of malaria and the clinical and immunological reactions induced by the malaria parasites presents many difficulties; for valid conclusions studies must be based on observations made under identical conditions of environment and exposure. Such situations are rare, and this is probably the reason why studies on this aspect of malariology are few and the conclusions drawn somewhat tentative and guarded.

The Negroes of West and Central Africa have evolved, throughout the ages, in continued confrontation with malaria; they have acquired practically total immunity to *Plasmodium vivax* and a high degree of tolerance to *P. falciparum.* Tolerance to the latter species implies no immunity to the parasite, but a high degree of tolerance to the effects of parasitization. Raichenow (*10*), in 1929, working among the Hausa of the Cameroons, reached the conclusion that toxin resistance was inherited but parasite resistance was acquired by continued exposure and reached its peak in early adulthood; thereafter, the parasite rate and the degree of parasitization remained low and more or less stable. Others in Africa have reached similar conclusions.

Holoendemic malaria is regarded as the highest expression of malaria endemicity: the spleen rate in children 2 to 12 years old is constantly above 75 per cent; the spleen rate and the size of the average enlarged spleen fall progressively with age, becoming low or negligible in the adult population. Mortality may be high in young children, but the adults enjoy good health so far as malaria is concerned. Holoendemic malaria is typical of Africa but has also been recorded in other rather restricted situations, as, for instance, among the aboriginals of northeastern India and in New Guinea. In the Western Hemisphere, however intense malaria transmission may be, so far as I know it has been recorded only among the Bush Negroes of Surinam—descendants of runaway slaves who have lived, for many generations, isolated in the Guyana forest, where other races, equally exposed, suffer severely and never acquire the high degree of adult tolerance to malaria infection that is characteristic of holoendemic malaria. This suggests that race may be a factor connected with holoendemicity.

Among the Bush Negroes, Swellengrebel and Van der Kuyp (*12*) found a very high *P. falciparum* rate in both children and adults; the mortality at all ages was low, however, and they were not much incommoded by the infection. Thus, in a village on the upper Surinam River, *P. falciparum* accounted for 75 per cent of the infections in children, and for 73 per cent in adults. The actual parasite rate averaged 70.5 per cent in children and 37 per cent in adults, yet during the twelve-year period from 1927 to 1939, the population of the village increased by 30 per cent, from 698 to 901, with an average annual death rate of 21, a birth rate of 39 per 1000 inhabitants, and infant mortality of 113 per 1000 births! Creole Negroes from the Surinam coastland, living in similar surroundings, showed the same or even higher infection rates and suffered severely.

The Amerindians of Surinam, living under the same conditions as the Bush Negroes, showed little evidence of tolerance to the infection. On the Wayombo River, in September 1939, 172 Indians were examined; *P. falciparum* accounted for 50 per cent of the infections in children (108 examined) and 61 per cent in adults (64 examined), and the spleen rates were 82 and 83 per cent respectively. Swellengrebel and Van der Kuyp found that

> The adult spleen rate and the average size of the enlarged adult spleen (2.2 Schuffner units), surpassing anything we encountered in Bush Negroes, are evidence of the lack of tolerance of these people to malaria parasites. We may add that a high spleen rate and a large size of the swollen spleen are peculiar of Indian malaria, as distinct from the Bush Negro and Creole malaria one meets everywhere in Surinam. . . . Besides a strong

> splenic reaction to the presence of malaria parasites in their blood, the Indians give evidence of their lack of tolerance by sickness and death. . . . The importance of Indians as carriers of malarial infection does not, like the Bush Negroes, consist in a tolerance to the parasite (manifesting itself by a moderate splenic reaction and a low morbidity and mortality), but in their seminomadic life which enables them to carry malignant malaria infection from the Bush to the Savannah.

American Negroes, descendants of slaves brought from Africa between the sixteenth and the nineteenth centuries, who have lived for generations under conditions of more or less moderate malaria endemicity, still enjoy some of the immunity built up by their ancestors. The North American Negro presents considerable resistance to provoked infection with *P. vivax* (*1*), and West Indian Negroes throughout the Caribbean, though equally susceptible to infection, on the average suffer much less and present lower spleen rates and smaller average enlarged spleens than other races living in the same environment (*2*).

In Guyana, people of six races have been living for many generations in the same general environment and often under the same conditions of exposure to malaria infection.[1] The Europeans (mainly of Portuguese descent) and the Chinese constitute small minorities and live mostly in Georgetown. The rural population of the coastlands comprises large numbers of Negroes and East Indians, and ideal conditions exist for comparative racial studies of all kinds. Up to 1946, when large-scale DDT spraying operations were introduced, malaria used to be stable and hyperendemic throughout the coast, from the Berbice Estuary to the Pomeroon River (*7*). Systematic surveys of schoolchildren were conducted between 1937 and 1946, separate records being kept for the two main races. The parasite rate showed no significant racial variation; splenic reaction in the Negroes was, however, decidedly and consistently more moderate, and decreased rapidly with age so that spleens of considerable size were found rarely in the older children and even less frequently in adults.

Among East Indians, on the contrary, the incidence and degree of splenic enlargement was much higher and was hardly affected by age. Clinical experience in sugar-estate hospitals showed that the majority of adults went through life afflicted by large—often very large—spleens and failed to come to terms with the infection; they suffered frequent attacks of fever, and anemia of some degree was practically universal. Mortality directly due to malaria was highest in children, but the tolerance established in adults by life-long exposure to infection was both low and unstable; at all ages, malaria and its sequelae continued to be major causes of death.

The 239,000 immigrants who came to Guyana from India between 1838 and 1917 originated mainly in Bengal, Bihar, Orissa, the Central Provinces, and Madras, where malaria is mostly, though not exclusively, hypoendemic or mesoendemic, and seasonal in its occurrence. This explains the lack of any inherited resistance to malaria and the severe reaction these immigrants suffered when they were transferred to the hyperendemic Guyana coastland.

Further inland, along the tidal course of the Berbice, Demerara, and other rivers, the autochthonous population is formed by Amerindians of the Arawak and Akawai tribes and by persons of mixed race, usually with Amerindian, European, and Negro blood in very variable proportion. There are few permanently settled Negroes, but a considerable fluctuating population of this race, mainly adult males, is employed in the mining and timber industries. At the time when malaria was hyperendemic in these districts, the Negroes came not only from the intensely malarial coast but also, and in high proportion, from the more mildly malarial Leeward and Windward Islands and from malaria-free Barbados. Whatever their origin, their reaction to malaria infection followed the same pattern: they contracted the infection soon after arrival, suffered an attack or two of fever, and after that enjoyed normal health and rarely returned for treatment.

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[1] According to the last census, the population of Guyana in 1960 totaled 560,406, comprising East Indians, 267,840; Negroes, 183,980; Mixed, 67,189; Amerindians, 25,450; Chinese, 4,074; Europeans, 3,218; and Others, 8,655.

In contrast, the mixed population born and bred in the area suffered severely and continuously, all ages being affected; mortality and morbidity were certainly graver in children, but the tolerance acquired by the adults was poor and very unstable. The Amerindians reacted in the same way as the mixed population, but even more severely; the largest spleens recorded, reaching the pubis and partly filling the pelvis, were seen in adults of this race.

Blackwater fever was not common in Guyana, in spite of the very high incidence of *P. falciparum;* it appeared at long intervals, during periods of malaria exacerbation, and tended to recur in some localities, with clustering of cases in certain neighborhoods, homes, and families. On the Demerara River between 1923 and 1931, 53 cases were recorded, mainly in 1926–1927, with the following racial incidence (*4*):

	Incidence of Blackwater Fever	
Race	*Per 1000 inhabitants*	*Per 1000 patients treated*
Negroes	2.67	19
East Indians	24.00	191
Amerindians	11.79	84
Mixed races	21.83	189

Nephrosis and chronic nephritis associated with *P. malariae* infection were frequent on the Demerara River. The racial incidence per 1000 patients during eight years was 20.4 in Negroes; 48.8 in East Indians; 33.6 in Amerindians; and 35.6 in mestizos. Children and young persons are more liable to blackwater and nephrosis than adults; thus the low incidence of these syndromes in Negroes might be due, to some extent, to the preponderance of adult males in this ethnic group. Ten years' experience on the Demerara River, in conclusion, showed that the Arawak and Akawai Indians of the area suffered severely from malaria and its complications and, like the East Indians of the coastlands, acquired only a very relative degree of tolerance to the infection, suffering from its effects at all ages.

In the far interior the population is widely scattered and can be estimated at 45,000 (1967), of whom approximately half are Amerindians belonging to the Wapishana, Macusi, Patamona, Akawai, Arakuna, and Carib tribes. On the Rupununi Savannahs and the Pakaraima Plateau, the inhabitants are almost exclusively Indians. Among them, malaria surveys have been few and irregular, and no systematic, long-term observations have been made; the same can be said of other medical studies. Prior to the introduction of control measures, malaria occurred throughout the year but transmission tended to be much more seasonal than on the coast, being related either to the May-August rains or to the floods of the larger rivers. Among the Indians of the lowland forest, spleen rates ranged from 40 to 60 per cent.

In some of the less accessible areas, the introduction of malaria appeared to be recent and possibly related to increased traffic with the coastlands. Thus, the savannahs of the Rupununi, which were difficult to reach before the advent of the airplane, were reported to be healthy up to 1930; the only Indians who suffered were those who had visited the coast, traveling down the Essequibo River or driving cattle through the Berbice trail. By 1938 (*5*), the infection had spread all over the savannahs, and more than 30 per cent of the children had enlarged spleens, the rates increasing with age. By 1943 (*6*), fears were entertained that the Makusi of the North Savannah were dying out. During a malariometric survey of this area, 424 Indians of all ages were examined; 68 per cent had either enlarged spleens, parasites, or both, and 46 per cent of the adults had large spleens, which in 20 per cent of cases extended below the transverse umbilical line. In the settlements situated nearest to the forest, 80 per cent of the inhabitants showed evidence of malaria. The lack of old people was very striking; only two were seen, who appeared to be about 60 years of age. This was reported to be a recent development; it was said that before malaria appeared on the savannah, in 1930, old and very old people were numerous. (One rancher who came to the area in 1922 had known two Makusi ancients who remembered quite well the British military expedition to Pirara in 1843.) During this 1943 survey, many settlements in the Indian reservation along the northern Kanuku Mountain foothills were found deserted and some of the houses burned, as is customary after the death of an inmate.

This high susceptibility of the Guyana Indians to malaria is without doubt one of the major causes of their decline over the past 150 years. Smallpox, measles, yellow fever, influenza, whooping cough, and tuberculosis have all been contributing factors, sometimes causing disastrous epidemics; none of these diseases, however, was as constant in its onslaught as malaria.

The early Dutch colonists sought the alliance of the Indians—particularly the Caribs, then a very numerous and warlike tribe—relying on them for the procurement of Indian slaves captured in raids on villages and Spanish mission settlements on the upper Cuyuni and Orinoco. Later, during the long years when African slaves were brought to the colony, both the Dutch and the British planters relied on Indian help in subduing slave rebellions and in the capture of fugitives. From 1778 to the end of slavery, annual official receptions were held in honor of the Indian chiefs. At the end of the eighteenth century, the Amerindian population appears to have been numerous and flourishing. The figures that have been given, however, are unreliable and differ widely; 50,000 would appear to be a reasonable guess. With the abolition of slavery, the Indians ceased to be of any interest to the colonists, and were left to themselves and neglected. Their numbers dwindled rapidly, and the Caribs, the most powerful and feared tribe, practically vanished; only a few survive at the present day on the upper Barama and Barima rivers. In 1848, after proper surveys, it was established that the Guyana Amerindians aggregated approximately 15,000, of whom only 7,000 were still in the aboriginal or semi-aboriginal state (*3*).

Systematic malaria-control operations were inaugurated in the interior in 1947; though eradication has not as yet been achieved, the incidence of the disease has been drastically reduced. The rapid increase that has followed in all the tribes is a clear indication of the disastrous effect of malaria on this population group over the past years; in 1968, their number is estimated at 32,000.

This high susceptibility and reactivity of the Amerindian to malaria is without doubt related to the recent introduction of the infection in the Western Hemisphere. It is generally accepted that malaria, as well as many other diseases, came to America with the discoverers, the conquistadors, and the hundreds of thousands of slaves brought from West Africa. In the Americas the new arrivals found, wherever they landed, native anopheles capable of acquiring and transmitting the disease; given the prolonged infectivity of malaria subjects, the lack of curative medicines at the time, and the wide diffusion of anopheles, it is not surprising that serious early outbreaks occurred among the invaders and that the infection spread rapidly to the highly receptive Indians.

In spite of tradition, it appears that even the Peruvian Indians were ignorant of the properties of cinchona bark. Richard Spruce, the botanist, spent fifteen years, from 1849 to 1864, on the Amazon, and in the cinchona forests on the slopes of Chimborazo in the Ecuadorean Andes (*11*). It was he who was responsible for the collection and shipment of seeds and young plants from which the cinchona plantations of India and Ceylon eventually originated. He found that the *cascarilleros* (Indians employed in the collection of cinchona bark) not only were completely ignorant of its medicinal properties but regarded his explanations with skepticism and suspicion, for they were convinced that the bark was used for the extraction of a valuable brown dye.

In equatorial America, altitude, in the Andean cordillera, has probably been the only barrier to the spread of malaria to even the most remote and isolated Indian communities. The Wai-Wai Indians are an entirely primitive tribe that has its main settlements in Brazil on the Mapueira River, which flows, through the Trombetas, to the Amazon, some 400 miles to the south. A few used to visit the headwaters of the Essequibo in Guyana. Their nearest neighbors are the Wapishana of the South Rupununi Savannah, some 70 miles to the northwest, through high rain forest. Cassava graters and feather ornaments have been traditional items of trade between these two tribes, but contacts are maintained by only a few individuals. For the last 15 years, a mission has been established at Kanashen, on the upper Essequibo, and it has attracted considerable numbers of Wai-Wai from across the border. In 1966 a group of 80 paid their first visit to the mission, after a journey of several weeks; of these, 14 were found infected with malaria, *P. falciparum, P. vivax,* and *P. malariae* all being identified.

So far I have dealt with the incidence of malaria and its effects on the American Indian. The Indian's way of life, however, also has important bearings on malaria epidemiology and even more so on the practical development of antimalaria operations.

Indian villages are small, with widely spaced houses. The larger buildings usually accommodate several families, each having its fireplace and allotted space. Completely isolated houses are frequent. The large villages to be found in the more civilized areas are due to missionary influence. The houses are made of materials that are easily procurable on site: round wood poles, bush ropes, palm leaves for roof thatching, bark and split palm trunks for walls and floors, and so forth. By community effort even the larger houses can be erected in the course of a few days, and a serviceable shelter may be put up in a matter of hours. This makes it easy for the Indians to move from one site to another for security, for better hunting or fishing, and, probably, for the purpose of escaping disease. As has been mentioned, houses in which a death has taken place are frequently abandoned and burned down. This instability of settlements has been recorded by most observers, and it is reasonable to expect that such a practice may help in the avoidance of malaria, which in the forest is eminently a place disease, prevailing in localities where conditions for abundant *A. darlingi* production exist.

The more permanent type of house constitutes the Indian's operational base, but, with his entire family, he spends a large part of his existence elsewhere—working on his farm, usually situated at a very considerable distance; on hunting or fishing expeditions; attending sprees; or just visiting his neighbors. In the more civilized areas, he may seek temporary employment—as a guide, a porter, or a boathand, bleeding balata (wild rubber), collecting Brazil nuts, and so on—and when so engaged he is usually accompanied by his family. When away from their permanent home, the Indians camp under temporary shelters, ranging from a few large palm leaves stuck into the ground to an open palm-thatched shed.

This great and continuous mobility of the Indian is without doubt a factor favoring the wide and rapid diffusion of malaria infection. In 1947 a sharp outbreak occurred among the Patamona Indians on the Potaro-Ireng watershed, on the Pakaraima Plateau, a mountainous, mainly forested region at an elevation of 1,000 to 2,000 feet above sea level (7). Infected persons were discovered scattered widely over some 500 square miles; all of them, however, had worked at, [illegible] had visited recently, a diamond placer, a[illegible] this was the only locality where transmission was actually occurring; a creek had been dammed and many gravel pits sunk in the valley bottom, and in these *A. darlingi* found ideal conditions for continued production. The migration of 80 Wai-Wai, of whom 14 were malaria parasite carriers, has already been mentioned.

These characteristics of the Indian way of life introduce serious difficulties in practical malaria control and may frustrate even the most efficient eradication program. In the sparsely inhabited interior, *A. darlingi,* in adaptation to the environment, is by no means as anthropophylic and endophylic as it is on the coast; it is widely distributed as a silvatic mosquito, and malaria transmission takes place not in villages and houses but at camping sites, on the farms, and

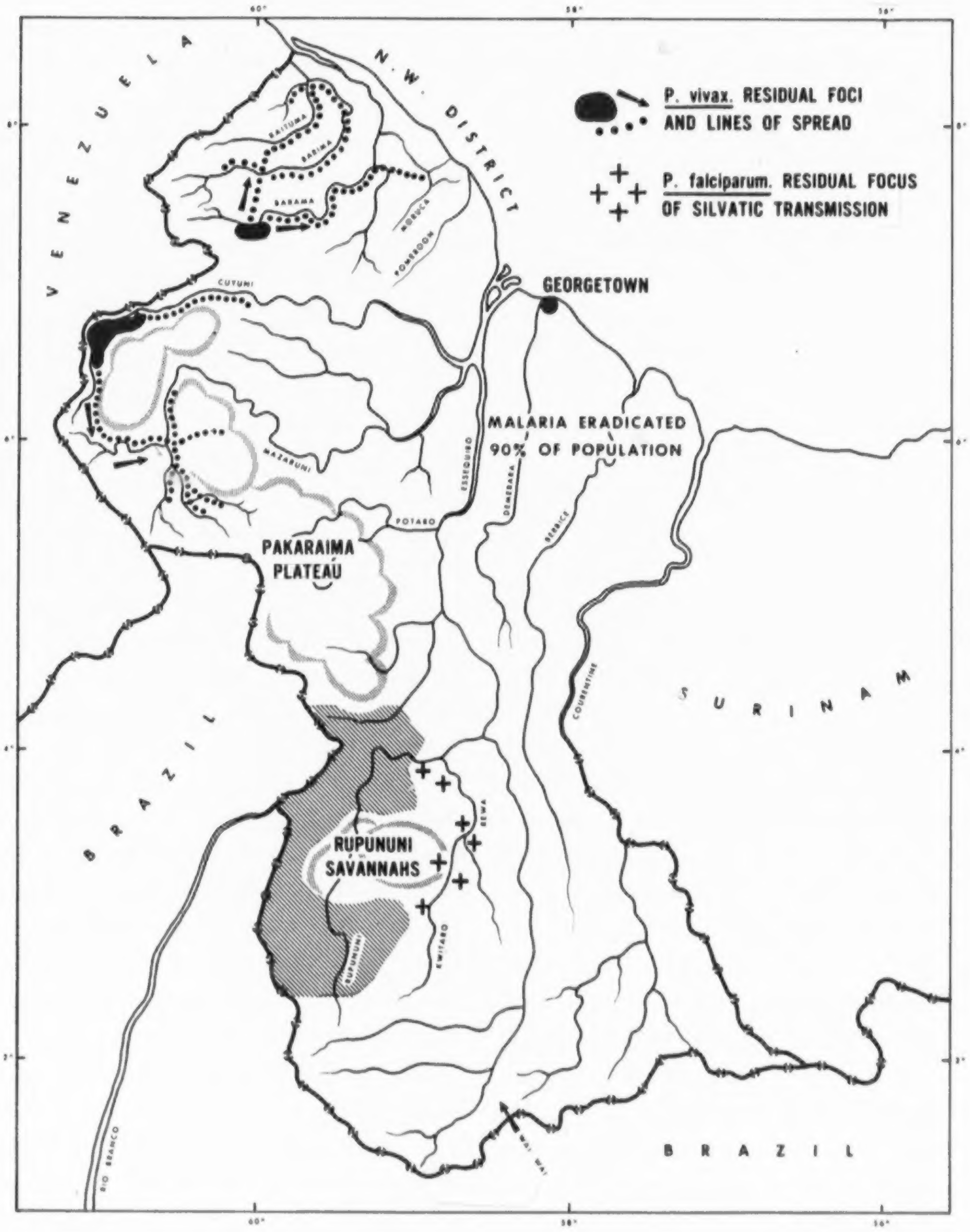
60°
58°
56°
8°
6°
4°
2°
VENEZUELA
N.W. DISTRICT
P. vivax. RESIDUAL FOCI AND LINES OF SPREAD
P. falciparum. RESIDUAL FOCUS OF SILVATIC TRANSMISSION
KAITUMA
BARIMA
BARAMA
MORUCA
POMEROON
CUYUNI
GEORGETOWN
MAZARUNI
ESSEQUIBO
DEMERARA
BERBICE
POTARO
MALARIA ERADICATED 90% OF POPULATION
PAKARAIMA PLATEAU
COURANTINE
SURINAM
BRAZIL
RUPUNUNI SAVANNAHS
REWA
KWITARO
RUPUNUNI
RIO BRANCO
WAI WAI
BRAZIL

FIGURE 1. Map of Guyana, showing residual *Plasmodium vivax* foci that survived 1961–1965 chloroquinized-salt campaign; lines of spread of infection by wandering Amerindians; small focus of *P. falciparum* in south, persisting among wild-rubber bleeders; and point of intrusion of primitive Wai-Wai Indians from Brazil. Elsewhere medicated salt and/or DDT house spraying have eradicated malaria or greatly reduced its incidence (see text).

In south, the campaign failed, following the introduction of a chloroquine-tolerant *P. falciparum* strain from Brazil in 1962. DDT house spraying on both sides of the border has interrupted transmission on the permanently inhabited Rupununi Savannahs, but a small focus of *P. falciparum,* transmitted by silvatic *A. darlingi,* persists among Amerindian balata-bleeders who camp, with their families, in the otherwise uninhabited Rewa-Kwitaro forest, from May to November. In all, 3,500 persons are involved, with an annual average of only 24 cases.

In the extreme south, the point of intrusion of primitive Wai-Wai Indians is indicated.

in the forest. The main villages can be sprayed with insecticides without excessive difficulty, but the existence of remote, isolated houses is very frequently unknown, and the systematic spraying of temporary shelters is not only impossible but futile, since they offer next to nothing in the way of sprayable surfaces. Under such conditions, insecticidal methods can reduce, but not eradicate, malaria, and their application is difficult and expensive. Systematic spraying of hammocks with DDT solution or emulsion is a useful practice, for the Indian always travels with his hammock and the insecticide residue resists occasional washing.

Mass chemotherapy, through systematic, periodic distribution of antimalarial drugs, is obviously impracticable, owing to the terrain, the widely scattered population, and its elusiveness. Even when a case of malaria has actually been discovered, treating it is a difficult matter; radical treatment of *P. falciparum* infection needs at least three days, and for *P. vivax* 14 days are required. Pinning down an Indian for regular daily dosage is no easy matter, and he usually fades into the forest in mid-treatment.

In Guyana, malaria, and its carrier *A. darlingi*, were eradicated from the coastlands and the more accessible and populated inland areas (93 per cent of the population) exclusively by DDT house spraying (*7*). In the far interior the results were by no means satisfactory, and malaria control only was established. This partial failure of DDT was due to the wide scattering of the population over an impervious terrain, its instability and mobility, and, above all, the silvatic adaptation of *A. darlingi* (*8*).

From January 1961 to December 1965 an attempt was made to eradicate these residual foci of malaria in the interior by means of chloroquinized salt (*9*); in order to assess fully the value of this new technique, DDT house spraying was suspended. This campaign covered an area of 42,000 square miles and a population of 48,500, mainly Amerindians. The campaign was conducted under strict administrative and scientific control; specific legislation was enacted making the use of chloroquinized salt compulsory in proclaimed areas and regulating its shipping, transportation, and sale in the interior. Epidemiological evaluation preceded, accompanied, and followed the campaign. In the areas in which chloroquinized salt was used, continued DDT operations, from 1947 to 1960, had greatly reduced the frequency of the disease; the reservoir of infection within the population was therefore relatively small.

In the North-West District and in the Pakaraima sectors, aggregating an area of 35,000 square miles and a 1964 population of 39,550, malaria disappeared within six months (the parasite rate in 1960 had been 8.3 per cent); thereafter, only four sporadic, cryptic cases of *P. vivax* infection were recorded up to April 1965, when the campaign was closed down.

Early in 1966, ten months after the cessation of all antimalaria measures in the area, a small focus of *P. vivax* infection was discovered among the Caribs of the upper Barama River—a very inaccessible locality that could receive chloroquinized salt only irregularly and probably in insufficient quantity, as no shops or other normal means of salt distribution exist. These Indians, traveling down the Barama, spread the infection to the Waini and Moruca rivers and, crossing overland, caused an outbreak at the manganese mines on the upper Barima and Kaituma rivers (see arrows at top of map). From there the infection spread rapidly to the lower course of these streams and to the Aruka.

In June and July the outbreak reached its peak, with 372 widely scattered cases. During 1966 a total of 643 cases were recorded on a total of 20,811 slides examined, out of a population of 16,850; *P. vivax* was the only parasite involved. The outbreak has been controlled by DDT house spraying and the reissue of chloroquinized salt, these techniques being used in combination for the first time. Only 15 cases were recorded in 1967 (9,116 slides examined) and only 3 from December 1967 to June 1968.

An identical incident occurred among the Akawai Indians of the Wenamu and upper Cuyuni, along the Venezuelan border, the first cases being discovered in April 1966, one year after the cessation of antimalaria operations. This again is a very inaccessible area, with a small population of Akawai Indians who can obtain salt and other supplies more easily from Venezuela than from the Guyana side; *P. vivax* was responsible for all cases discovered. Indians crossing overland from the Wenamu to the Kamerang, an affluent of the Mazaruni, spread the infection to the upper Mazaruni and its tributaries, a sparsely inhabited area with an estimated population of 3,000 Indians and diamond miners. During 1966, 280 cases were discovered in this sector. Many infected persons reached the coastlands, but were intercepted by systematic examination of plane passengers from the interior. DDT house spraying and chloroquinized salt (issued free to Indians) have brought the situation under control, though progress has been slow, owing to the very difficult terrain.

It is important to note that both these recrudescences were due exclusively to *P. vivax;* the 1961–1965 medicated-salt campaign, even in remote and poorly supplied areas, appears to have achieved the eradication of both *P. falciparum* and *P. malariae,* which before the introduction of chloroquinized salt accounted for 57 and 15 per cent, respectively, of the infections.

Among the 10,000 Makusi and Wapishana Indians who inhabit the savannahs of the Rupununi, close to the Brazilian border, chloroquinized salt failed, after a favorable start during 1961, owing to the introduction of a chloroquine-tolerant *P. falciparum* strain from the neighboring Rio Branco Territory (*9*). This new strain spread rapidly over the whole area, even where chloroquinized-salt coverage was adequate. DDT house spraying, interrupted since 1960, was reintroduced in 1962, and as soon as systematic spraying operations were established by the Brazilians across the border, beginning in January 1965, malaria transmission on the savannahs was interrupted. Silvatic *A. darlingi* still persists in the surrounding forest, however, and occasional cases of *P. falciparum* malaria continue to occur, but exclusively among Indians engaged in bleeding balata, who camp in the forest during the rainy season, from April to November. Among 700 bleeders and their dependents (estimated at 2,800), 24 cases were discovered in 1966 and 22 in 1967.

For the 1968 season it is proposed to issue to all bleeders, along with the other rations they receive, 20 pounds of salt medicated with chloroquine (0.43 per cent chloroquine base) and pyrimethamine (0.043 per cent), and, in addition, to give to every individual who can be "caught" before leaving for the forest a two-day treatment with a combination of Fanasil (Sulfurthomidine) and pyrimethamine. The result of this new approach will not be known till the end of the year.

These various developments in the course of the Guyana campaign illustrate clearly the peculiar difficulties encountered in malaria control and eradication operations among the Amerindians. As I have said, in the early planning of the many campaigns that have been undertaken of late years throughout tropical America, little importance was attached to the specific problem of malaria in the Amerindian, priority being given to the larger, more accessible communities, which were economically more important and politically more vocal. Today, with eradication or, more frequently, control established over wide areas, the problem of malaria among the Indians assumes a new dimension, for it is evident that eradication will remain a dream until means have been discovered to extend control to even the most remote of Indian settlements. I have shown how rapidly and widely malaria can be broadcast from extremely limited foci, by wandering or migrating Indians.

Of the preventive techniques known at present, medicated salt appears to be the most promising, since it can find its own way far beyond the reach of organized public health agencies. We do not yet possess, however, the ideal drug for this purpose. Those in use today, among other shortcomings, fail to act on certain parasite strains and may thus bring about the emergence and favor the diffusion of drug-tolerant or drug-resistant infections, introducing therapeutic problems which may be serious.

I have shown how, in Guyana, chloroquinized salt was completely successful against all forms of native malaria parasite, only failing where and when a chloroquine-tolerant strain was introduced from Brazil. Our campaign, it should be emphasized, was inaugurated at a stage when the reservoir of infection had been greatly depleted by DDT spraying. Under such conditions, the probabilities of the presence of resistant strains were much reduced. With the drugs at present available, we believe that medicated salt should not be used under conditions of high endemicity.

Indians still in the aboriginal stage of civilization are not acquainted with commercial salt and manufacture salt substitutes by washing certain soils or from the ash of certain palms. Natural deposits of salt are not likely to occur throughout the vast equatorial rain forest. On the other hand, civilized and semicivilized Indians take to the use of commercial salt very readily, as in it they find not only a means for seasoning their food but a valuable preservative for the fish and game on which they subsist and which tend to abound in certain seasons and not in others; the traditional method of preservation employed is smoking. The entirely primitive Wai-Wai Indians, for instance, who reach Kanashen Mission from the depth of the Brazilian virgin forest, take to the use of salt with great readiness as soon as it is made available.

Obviously, spreading the use of salt to the more remote tribes will take years, and the same can be said for eradicating malaria where primitive Indians exist; at the present stage of malariological knowledge and antimalaria technique, it appears likely that treatment and prevention may be extended more easily to the primitive and semiprimitive Indians through the established channels of intertribal barter trade, in a bag of salt, than through a spray gun or a bottle of tablets. For this purpose, however, more effective drugs for salt medication have to be found.

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FOOD AND NUTRITION OF THE MAYA BEFORE THE CONQUEST AND AT THE PRESENT TIME[1]

Moisés Béhar

". . . and grinding then the yellow and the white ears of corn, the goddess Ixmucané prepared nine beverages, and from this food came strength and stoutness and with it they created the muscle and vigor of man. . . ."

"Their flesh was made from yellow and from white corn; from ground and kneaded corn were moulded the arms and legs of man. Only damp, ground corn went into the flesh of our fathers. . . ."

A great civilization developed in Middle America on the basis of corn. The above quotations from the *Popol Vuh,* the sacred book of the ancient Quiché Maya, emphasize the importance ascribed to this staple grain by the Mayan populations. Nevertheless, our present knowledge of nutrition has demonstrated that corn, if not properly supplemented (*16*), is nutritionally very poor as a staple. As in most of the cereal grains, its protein concentration is low; in addition, corn proteins have a very low biological value. They are deficient in at least two of the essential amino acids: lysine and tryptophane (*5*). Furthermore, the deficiency of tryptophane in corn, plus a low concentration and/or availability of niacin, favor the development of pellagra in those whose diet is based on it (*10*).

It must therefore be asked how an important civilization like the Mayan could develop with such a great dependence on corn as its staple food; and why the present-day descendants of the Mayas have serious nutritional problems, also because of their dependence on corn.

In this paper I shall try to review our unfortunately small store of information on the dietary practices and nutritional status of the Mayan populations before the Spanish conquest of this region. I shall examine the same conditions in their direct descendants, the Guatemalan Indians of today, and attempt to determine the main changes that have come to pass. It is my hope that, in so doing, some general principles applicable not only to this particular group of American Indians but to others as well will emerge. This seems to me more useful than a purely descriptive paper on the nutritional problems now afflicting the American Indian groups.

Dietary practices and nutritional status of the pre-Columbian Mayan Indians

Corn, prepared in many different ways, was eaten at every meal, every day, as the most important food. In addition, the Mayas cultivated and consumed beans, vegetables, fruits, roots, tubers, cacao, and spices (*15*). Animal products, mainly meat and to a much lesser extent eggs, were apparently not a regular item in their dietary; they were consumed sporadically and on special occasions, and were obtained mainly by hunting. Very significant in this regard is the existence, in a Mayan dialect of the Guatemalan highlands, of a special word, *tcibex,* for "to eat meat," as distinct from the general word *lobex* meaning "to eat" (*14*). Only the groups living on the coasts consumed fish. Milk was not used at all.

Our present knowledge of nutrition indicates that, with such a diet, it is difficult to satisfy the requirements for at least three important nutrients: protein, calcium, and niacin. The deficiency of calcium and niacin was corrected by the way in which corn was processed. For the preparation of *tortillas* and other corn products, the grain was first soaked overnight in lime water and then cooked in the same water (the purpose was to remove the husk). The result was a product very rich in calcium, for which the Mayas had no other adequate dietary sources. This method also yielded a higher concentration of tryptophane, in relation to the other amino acids, and more available niacin (*13*), thus preventing pellagra. We can only speculate on how this practice was adopted, but it is now evident that it was indispensable for their survival.

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We are still left with the problem of how they satisfied their nutritional requirements for protein. As I have said, the main drawback of corn proteins is that they are deficient in some essential amino acids, particularly lysine and tryptophane. However, an amount of 39 grams of corn protein a day, which satisfies the average protein requirement in an adult male weighing 65 kilograms (*17*), does contain the needed amounts of all essential amino acids (*12*). This amount of protein is provided by about 370 g of corn, which is not an unreasonable quantity for a man to consume per day—in fact, Guatemalan Indians now consume around 500. In spite of the low biological value and low concentration of proteins in corn, it is therefore possible for adults to satisfy their protein needs with this grain as the main staple, if they consume enough of it.

But the situation is completely different for small children, who have relatively higher protein requirements and in whom the need for essential amino acids is also greater. A two-year-old child, for instance, would need about 18 g of protein a day to satisfy his nitrogen requirements if the protein has an ideal composition, with a biological value of 100 per cent; in other words, if it is fully utilized (*17*). With corn as the source, the child would need 36 g of protein, because the biological value of corn protein is about 50 per cent (*4*). This means that he would have to consume about 450 g of corn a day, or about 800 g of tortillas, to satisfy his protein needs. Not only is this physically impossible but, in addition, the child would be receiving many more calories than he needs.

Of course, the Mayas did consume other foods besides corn. From what we know, the other important items in the regular daily diet were beans, fruits and vegetables, and roots and tubers. Beans are a more concentrated source of protein, but the remaining food items, although they contribute other needed nutrients, reduce the over-all concentration of protein in the diet. The situation would again be one in which adults can satisfy their nutritional needs but small children cannot.

I have mentioned that the Mayas did not consume milk, and that other animal products (meat and eggs) were only eaten irregularly. It is also very unlikely that they fed meat to infants and small children, for they had not developed the required processing facilities. There was only one solution, and they applied it—prolonged lactation. The information now available indicates, in fact, that Mayan mothers breast-fed their children for at least three years and frequently even longer (*15, p. 44*).

Our present knowledge indicates that breast milk can satisfy all the nutritional requirements of a baby up to approximately six months of age. It is probable that from about that age Mayan mothers started to give their children what the rest of the family ate but continued to feed them breast milk as an indispensable protein supplement. By the time they were completely weaned, after the age of three, they were able to eat enough of the family food to satisfy their nutritional requirements without the use of any special product, such as milk in the case of Western civilization. It is also probable that during this transitional period, from about six months to three or four years of age, they did not grow to their full genetic potential. This could be the main reason for the small size of the adults, which is well documented. This reduction in body size, a consequence of environmental conditions and particularly of nutrient availability, can also, I believe, be considered a mechanism of adaptation to those environmental conditions. In fact, with reduction in body size resulting in a similar reduction in nutrient requirements, the Mayas were better able to meet these requirements with the available foods.

Unfortunately, we have no information on the health conditions of children in those populations. Data on infant and early childhood mortality could be particularly illustrative. The information available indicates that, in general, the children were healthy. De Landa (*15*) says:

> They grew wonderfully handsome and fat during the first two years. Later, their skin waxed dark with continuous bathing by mothers and many suns; they were nonetheless bonny and mischievous throughout childhood, never ceasing to wander about carrying bows and arrows and frolicking among themselves and thus they grew until they began to adopt the ways of the youths and to acquire a more exalted self-concept, and to leave behind the things of childhood.

We cannot say the same about the majority of preschool children of Mayan descent today; they are usually apathetic and very often sick.

Another important deduction that can be made from the information on late weaning concerns birth spacing. The mothers probably had at least three years between one birth and the next. To what extent prolonged lactation contributed to this situation we still do not know. However, it was definitely very convenient, because only in this way could the mothers breast-feed their children as required. Accurate information on the nutritional condition of pre-Columbian Mayan adults is also nonexistent, but all known facts indicate that, although small, they were in general strong and healthy. We can thus conclude that, in terms of nutrition, they did not have serious problems and were well adapted to their environmental circumstances and degree of cultural and technological development. They did, however, undergo acute crises, and there is information on the famines they suffered when natural disasters (droughts or locust invasions) interfered with their corn crops.

Dietary practices and nutritional condition of the Guatemalan Indians of today

The dietary practices of the Guatemalan Indians of our time have not changed very much from those of their pre-Columbian ancestors. The changes that have occurred, however, have gradually brought about a deterioration in their diet and have upset the balance under which they lived.

Corn, processed in the same way, is still the main staple. It provides up to 80 per cent of the calories and 70 per cent of the proteins in the diet of the adult population (*9*). Beans, vegetables, and fruits complement the basic diet (*8*). These products, however, now have a market value for which the more affluent non-Indian groups in the population are better able to compete. This is even more true of animal products. Wild animals have practically disappeared, and hunting is no longer an important means of obtaining precious foods. Domestic animals are now the only important source of animal products, and the market for them is very competitive. The majority of the Indian families raise some animals in their homes, mainly poultry and pigs. But these are more a source of badly needed cash than of food. They and their products are consumed only on very rare occasions; most of the time, they are sold. The meat most frequently consumed is beef, which has to be obtained from the market and therefore is one of the luxury items for which cash is needed.

Not only have the Indians lost their old sources of food in the wild forests, but also the amount of land available to them for cultivation has been drastically reduced. Before the Conquest, they had all the land they needed to produce their corn. Although the method of cultivation they used cannot be considered very efficient today, its relative inadequacy was compensated for by the availability of sufficient land for a rotation system. They did not need to produce cash crops, which now compete with food crops for the best lands.

Under present-day circumstances, the Indians are frequently forced to buy even their corn, which they had always produced before. Owing to their poverty, this greater dependence on the market to satisfy food needs has caused their diet to deteriorate. Another change in the adult diet is the introduction of coffee, which has replaced the more nutritious corn and chocolate beverages they used to drink. Distilled alcoholic beverages have also become part of their consumption habits, with deleterious effects from the nutritional and even more from the economic standpoint.

Changes in dietary practices concerning small children are even more important, and these have been mainly the effect of cultural influences. In the first place, the period of weaning has been shortened. In pure Guatemalan Indians, prolonged lactation is still essential to children's survival. Complete weaning in these populations now takes place between the ages of about two and two and a half years; this time, already shorter than it used to be before the Conquest, is being progressively reduced as the process of integration into the *ladino* or Western culture advances (*11*). This situation is very dangerous and has caused acute nutritional problems, since the populations adopting earlier weaning are not as a rule prepared culturally and economically to feed their children properly during the critical post-weaning period. Their general ignorance in matters of hygiene, and the lack of the most elementary physical facilities in their homes, renders the use of milk very dangerous. Most of them, in any case, cannot afford to feed milk to their children.

The situation is further aggravated by the introduction, by foreign civilizations, of refined products with a much lower nutritive value than the foods they replace. This is particularly true and deleterious for the child during the post-weaning period. An example of this problem is the pure starch preparations made from corn or manioc that are often used nowadays in gruels for small children, instead of the more nutritious whole-corn gruels of old.

The Guatemalan Indians of today thus seem to be in a less favorable balance with their environment than their ancestors were, and their dietary practices have deteriorated. Food intake at the present time is barely adequate for adults and frequently inadequate for children. As a result, environmental stresses, particularly the heavy burden of frequent infections in early childhood, have caused poor nutritional conditions for the general population, with frequent and severe cases of nutritional deficiencies found particularly in early childhood. Briefly, the nutritional problems now affecting the Guatemalan Indians are as follows:

The most important by far is protein-calorie malnutrition (*19*). As has been indicated, it is the result of the synergistic effect of an insufficient and inadequate dietary intake and frequent infectious episodes, particularly diarrheal processes and the common diseases of childhood (*3*). This condition contributes very significantly to the high mortality rates in infancy and early childhood (*2*). It causes a relatively high prevalence of severe cases of protein-calorie malnutrition (kwashiorkor and marasmus) (*1*), which are usually fatal if not properly treated in a hospital. Even more important from the public health standpoint are the chronic, subclinical forms of malnutrition, which usually go unrecognized. These affect a large majority of the population, both children and adults (*18*). The consequences of this condition are not yet clear, but there is information suggesting that it may interfere significantly with mental development in children (*6*) and with work performance in adults (*20*). If these observations are confirmed —and studies are now under way to test the hypothesis—it is easy to understand the tremendous importance of the problem in terms of the general well-being of these populations and of their participation in the social and economic development of their societies and countries.

Among the other nutritional problems may be mentioned vitamin A deficiency, primarily the result of diets in which the sources of this vitamin are vegetable products with low biological activity consumed in insufficient amounts (*8*). This deficiency results in problems as serious as keratomalacia, which ends in total blindness during early childhood. Riboflavin deficiency is among the most prevalent of the vitamin deficiencies (*8*). It is closely interrelated with protein deficiency, since the adequate dietary sources of the two are frequently the same. Mucosal and cutaneous lesions characteristic of this deficiency are frequently seen. Its possible general effects are not clearly understood.

Nutritional anemias are also highly prevalent; the deficiencies of iron and folic acid are the factors most frequently responsible, but again protein malnutrition contributes to their development (*21*). Anemias may also be important in restricting work capacity. Endemic goiter used to be highly prevalent in Guatemala, but fortunately it has now been controlled by means of salt iodization (*7*).

The nutritional problems herein summarized do not affect the Indian population in Guatemala specifically or preferentially. They are associated with low educational and economic levels and poor sanitary conditions. There are non-Indian populations in similar conditions, but practically all the Indian populations fall into this category. Their difficulty is one of proper integration into and adaptation to new ecological and cultural conditions and social structure.

In my opinion, the situation described in relation to the Mayan Indians, and the changes that have come to pass, are applicable to a lesser or greater extent to other Indian groups now living in touch with Western civilization in the Latin American countries. On the other hand, many Indian groups still living in isolation from Western culture and from the present Latin American societies have less serious nutritional problems; some of them seem to be living under excellent conditions in this respect. These groups are still very dependent on hunting and fishing to satisfy their food needs, and still follow the "primitive" practice of raising their children at the mother's breast for as long as possible. They do not have at their disposal the "benefits" of civilization, such as the practice of bottle feeding and very inadequate weaning foods. We must consider the situation of these groups of primitive Indians as they become incorporated into our societies, with the hope that we will find means to prevent or minimize the nutritional deterioration to which they may be exposed in the process of integration.

Summary

An analysis of the dietary pattern and the nutritional condition of the Mayan populations before the Conquest and at the present time indicates that, although corn constitutes the main staple for both population groups, the pre-Columbian Mayas were probably better adapted to ecological conditions. The availability of game, greater availability of land for cultivation, the absence of competitive markets, prolonged lactation of infants, and the lack of industrialized products were among the factors that brought about a better diet for the Indians at that time. The sociocultural and economic changes that have taken place since the Conquest have worsened the diet of the present Indians and resulted in serious nutritional problems, among which protein deficiency—particularly in small children—is the most important. This experience should be considered both in trying to solve the nutritional problems of this and other American Indian groups now living with populations of Western culture and in preventing similar changes that could occur as the still primitive and isolated Indian groups are incorporated into modern Latin American culture.

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IODINE DEFICIENCY WITHOUT GOITER IN ISOLATED YANOMAMA INDIANS: PRELIMINARY NOTE

René Rivière, Dominique Comar, Mentech Colonomos, Jean Desenne, and Marcel Roche[1]

Roche *et al.* (*10*) showed that, in a region of endemic goiter, subjects without as well as those with goiter had an elevated thyroid radioactive iodine uptake. A similar finding was reported in New Guinea by Choufoer, Van Rhijn, Kassenaar, and Querido (*1*). In an isolated primitive Venezuelan Indian community, Roche (*9*) demonstrated a generally elevated thyroid uptake without goiter; and this was again described in the eastern region of the Congo Republic by Delange, Thilly, and Ermans (*3*).

In the present preliminary note, further quantitative aspects of thyroid metabolism in a Yanomama group from the upper Orinoco region are presented.

Material and methods

The area chosen for study lies roughly between the Ocamo and Mavaca rivers on the Orinoco (Figure 1), in the extreme south of Venezuela. It is reached by plane to La Esmeralda—an old mission site visited by Humboldt (*7*) in 1800—and upstream from there by fast river boat in about four hours. Two expeditions were carried out.

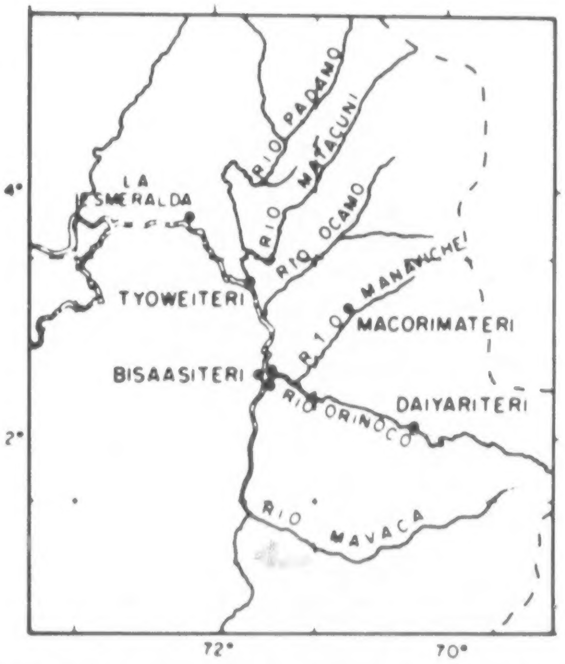

FIGURE 1. Area selected for study of thyroid metabolism in Yanomama Indians (see page 77 for map of Venezuela)

During a preliminary one in February 1962, thyroid iodine uptake was determined on 41 Yanomama Indians living in Bisaasiteri, at the confluence of the Orinoco and the Mavaca. All the subjects studied were adequately nourished, in good health, and with thyroids that were either not palpable or barely palpable. A Catholic and a Protestant mission were established at the site, but the Indians still lived in wide-open thatch huts, ate their own food, and went completely naked.

Another expedition was carried out in January, February, and March 1968 to Bisaasiteri and to the neighboring Ocamo River (Tyoweiteri). The living conditions of the Indians had changed, although they remained very primitive. They no longer lived in the characteristic huts, but rather in closed dwellings; they occasionally received food and medicines from the missions. A third group, from Macorimateri on the Manaviche river, was also contacted this time; this group, unlike the first two, was still culturally isolated in all respects.

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[1] Presented by Dr. Roche.

On the second expedition, 56 adult subjects—44 male and 12 female—were studied. They differed from those studied on the first expedition. They were in apparently good clinical nutritional state, of short stature (males 1.57 m and 52 kg, females 1.44 m and 45 kg, on the average) and without any clinical evidence of thyroid dysfunction. In 45 of them the thyroid was not palpable; in 10 it was palpable but estimated to be of normal size. In one subject (a male) there was a small diffuse goiter, estimated to weigh around 50 g.

Twenty-four-hour thyroid uptake was determined after the oral or intravenous administration of 5 to 30 μc of ^{131}I, in the manner recommended by the International Atomic Energy Agency (*12*). A figure on the uptake of the first group has been published (*6*). Kinetic studies are being continued after the oral administration of 100 μc of ^{131}I and will be reported later, as will the effect of TSH and of methimazole and other measurements.

Radioactivity was measured on the site of the study; the various iodinated fractions of plasma were separated on ion exchange resins and measured separately. Measurements of urinary ^{127}I and creatinine were performed on single urinary samples because of the practical impossibility of obtaining reliable 24-hour samples.

The blood, urine, and river drinking water samples were refrigerated around 4° C when indicated and sent by plane to the central laboratory in Caracas. Stable iodine was measured by activation analysis in the Nuclear Chemistry Department of the Venezuelan Institute for Scientific Research and at the Service Hospitalier Frédéric Joliot in France (*2*). The iodine/creatinine ratio was measured to give an idea of the daily urinary iodine excretion. The average 24-hour creatinine excretion was assumed to be 1.6 g for men and 1.0 g for women (*5, 8*).

Results

Figure 2 shows the thyroid iodine uptake in Bisaasiteri in 1962 (left) and the uptake in the combined populations of Bisaasiteri, Tyoweiteri, and Macorimateri in 1968. Table 1 shows data on iodine metabolism. The iodine content of river water was 3.2 μg/liter (average of six samples).

Discussion

As Figure 2 shows, the thyroid iodine uptake is higher in all the subjects studied in 1962. In 1968, although many uptakes are still high, this time five individuals have uptakes below 40 per cent. It is presumed that the lowered uptakes are due to the introduction of foreign food and medications; none of the uptakes in the more isolated group were low, and the average in that group was higher than in the other two (Table 1).

The urinary iodine/creatinine ratios (Table 1) are low compared to normal values (*5*). The total 24-hour iodine excretion can only be indirectly evaluated. The values obtained are approximations, since urinary excretion of iodine is not uniform throughout the day and since 24-hour urinary creatinine is not an accurately fixed value. As a matter of fact, creatinine excretion is related to muscle mass and in these short, thin Indians is likely to be less than is taken here as normal (*4*.) In a group of Makiritare In-

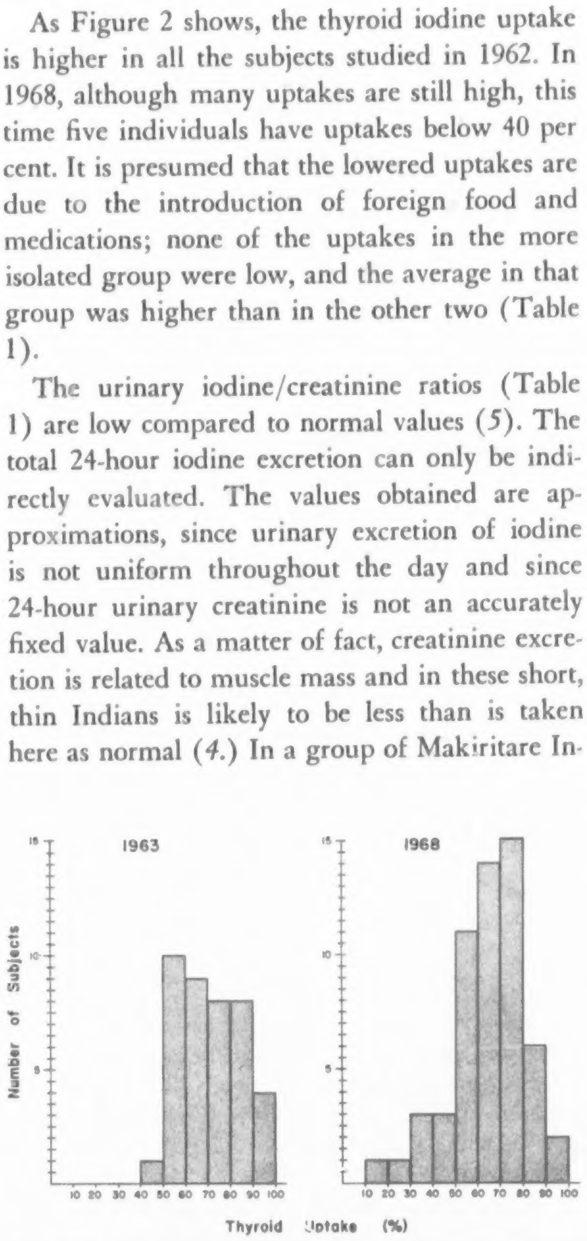

FIGURE 2. Frequency distribution of iodine^{131} thyroid uptake of Yanamama Indians

TABLE 1. Iodine metabolism in isolated Venezuelan Indians (average, standard deviation, range, and number of subjects)

SITE	24-HOUR THYROID ^{131}I UPTAKE (% OF DOSE)	^{127}IODINE (μg)/ CREATININE (g) RATIO	PLASMA THYROXINE ^{127}I (μg/100 CC)	URINARY ^{127}I (μg/DAY)	MAXIMUM ^{125}I PLASMA THYROXIN (%/LITER)
Bisaasiteri	64.3 ±7.4 36.0–81.3 (26)	29.1 ±5.2 10.0-104.0 (35)	4.42 ±0.53 2.8–5.7 (22)	42.6 ±7.8 15.0–166.0 (35)	0.445 ±0.107 0.194–1.100 (18)
Tyoweiteri	61.1 ±10.3 48.6–70.0 (10)	—	—	—	0.679 ±0.221 0.207–1.450 (10)
Macorimateri	74.1 ±12.4 32.9–93.0 (12)	19.1 ±4.0 30.0–70.0 (10)	5.07 ±0.84 2.6–7.4 (12)	30.5 ±6.4 16.0–48.0 (10)	—

dians, at the Ventuari River, Roche, Perinetti, and Barbeito (*11*) found a 24-hour iodine excretion of 21.2 μg/day with a mode between 10 and 20 μg. Iodine in the drinking water was low in the Orinoco, although not as low as in the Ventuari, where, by a different method, 0.2 to 0.6 μg of iodine was found.

Plasma thyroid ^{127}I was normal in both of the groups studied, which is in accord with the observation that these subjects are clinically euthyroid.

The high thyroid iodine uptake and the low urinary ^{127}I excretion are comparable to those found in a goitrous Andean region of Venezuela (Rivière, Camuzzini, Comar, and Roche, to be published) and indicate that these goiter-free Indians are indeed iodine-deficient. It again appears evident that goiter may fail to occur even though, as is likely in this case, iodine deficiency has been present through many generations. What other factor or factors are needed remains in question.

Summary

A study of iodine metabolism was carried out in 48 Indians from the Yanomama tribe living near the confluence of the Orinoco and Mavaca rivers in Venezuela. Elevated thyroid iodine uptake, low urinary iodine/creatinine ratio, normal plasma thyroxine, and apparently normal clinical thyroid status were demonstrated, in the absence of goiter. It is suggested that iodine lack is a necessary condition for the formation of endemic goiter.

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STUDY OF ENDEMIC GOITER IN THE AMERICAN INDIAN[1]

José Barzelatto and Edmundo Covarrubias[2]

The existence of endemic goiter in America can be traced back to pre-Columbian times (*5, 43*), although the opposite view can be sustained (*14, 15*). Endemic goiter areas have been demonstrated during the last decades in practically every country in the Americas. From the review published by Kelly and Snedden in 1960 (*21*), it can be estimated that at least 20 per cent of the population is affected by goiter. From epidemiological surveys comprising in some cases huge samples, it can be inferred that all races are affected. Unfortunately, there is no precision about racial distribution in most of the studies; present-day American Indians are mixed in different degrees and have been included in variable proportions. Those Indians who live in primitive conditions and are presumably less mixed have been the subjects of only a few specific goiter surveys (*35, 46*).

Iodine deficiency in the populations of endemic goiter areas in America, as judged by urinary excretion, has been found in practically every case where it has been looked for (*2, 17, 18, 19, 24, 26, 36, 41*). This is not surprising, since except for the nitrate deposits in northern Chile (*20*) iodine is not abundant in the surface of this hemisphere as it is in the rest of the world. Wherever the diet has been supplemented with iodine, the incidence of endemic goiter has been greatly reduced; well-documented examples are those of the United States (*22*), Mexico (*40*), Guatemala (*39*), Colombia (*6*), and Argentina (*33*). Iodine prophylaxis programs are being increased in number and effectiveness in America and will soon influence goiter epidemiology, at least among Indians more closely integrated with the surrounding society.

The idea that endemic goiter reflects only the adaptation of man to iodine deficiency can no longer be sustained. As in other parts of the world, it is contradicted in America by such examples as the finding of groups of Indians without goiter who were iodine-deficient (*37*) and the persisting development of endemic goiter in children born in areas where effective iodine prophylaxis was under way (*13*). Natural goitrogens occurring in food have been suspected of playing a role in the pathogenesis of some endemic goiter (*11, 23, 32, 34*). An example in our continent is the piñon or nut of the *Araucaria araucana*, a pine tree abundant in south-central Chile (*42*).

Contrasting with the situation in sporadic goiter, genetic mechanisms have not been well documented in the pathogenesis of endemic goiter. Enzymatic defects have not been demonstrated in its etiology. A concentration of endemic goiter cases within families has been reported in America (*16, 25*) and elsewhere (*7, 27*). A positive association of nodular goiter with the distribution of nontasters of phenylthiocarbamide (PTC) has been reported in a general mixed population in Brazil (*28*). However, the search for this association among

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[2] Presented by Dr. Barzelatto.

American Indians is rendered difficult by the small proportion of nontasters usually found among them, as was the case in a study of the Pewenche (*10*).

Another aspect to be considered is the problem of endemic goiter variability. The anatomic and physiological characteristics of goiter may vary in a single individual during his lifetime; other levels of variation are to be defined among the members of a given community and among different populations. These three types of variability make it a hard task to compare results of different authors, especially because there is still no general agreement on definitions. Some methodological limitations should also be borne in mind, such as subjective differences when not all individuals are examined by the same person. Even if these are overcome, difficulties in classification persist, such as estimating the size of a goiter in the presence of retrosternal extension and the error induced by missing goiters that have become totally retrosternal.

The impact of endemic goiter in public health is another matter of debate. Severe endemic goiter is associated with endemic cretinism, deaf-mutism and neurological defects, but the mechanisms involved are not clearly understood. There are also no adequate studies of mortality and fertility differentials in comparable populations with and without endemic goiter, or in individuals within an endemic goiter area. In both cases fruitful information may be obtained from looking into relations with endemic goiter variability.

Comparative studies among different groups of American Indians could provide an excellent opportunity to study the pathogenesis of endemic goiter. They presumably have very different genetic endowment, and also are influenced by different environments. The interplay of all these factors in the genesis of endemic goiter is probably unique in each population; hence studies in depth of endemic goiter in certain communities may be scientifically rewarding, besides providing the basis for rational prophylaxis programs.

Endemic goiter in Pedregoso

In accordance with the foregoing line of thought, we have been involved for the last five years in a study of endemic goiter on a Pewenche Indian reservation in Chile. Some of our findings will provide a good basis for discussion of these ideas.

Pedregoso is a geographically isolated Andean valley at an altitude of 1,310 meters, with a cold climate. According to a 1965 census, 592 Pewenches live there, by relatively primitive agriculture and some cattle raising. Piñon is a standard item in their diet. The available genetic data show that these Indians are less mixed than other groups of the Araucanian complex to which they belong (*8, 10, 29, 30, 38*) and constitute a highly inbred population in which a quarter of the marriages are consanguineous (*10*).

Endemic goiter in Pedregoso was found to have a prevalence of 66.8 per cent in a sample comprising 81 per cent of the total population (the remaining 19 per cent, mainly young people, were not examined in our house-to-house survey because for one reason or another they were away from home). The distribution according to sex and age is shown in Table 1, which also gives the frequencies of clinically nodular and large goiters (including those estimated to weigh 60 grams or more and all those with retrosternal extensions). As in most other studies, women were found more frequently goitrous at all ages than men. The fact that the frequency does not diminish after puberty and the finding of nodular goiters in the first decades of life are indications of the severity of the endemic. On the other hand, huge goiters were exceptional and only one cretin and no deaf-mutes were found.

The first problem to be considered is whether the increases in size and in nodularity with age are independent effects. Nodularity increases with age in both sexes, appearing earlier among women (Table 2). Large goiters are more frequently nodular than small ones with advance in age, but some individuals have small nodular goiters in the first decades. It is obvious from

TABLE 1. Goiter in Pedregoso

AGE (YEARS)	MEN					WOMEN				
	EXAMINED		GOITROUS			EXAMINED		GOITROUS		
	N	% GOITROUS	N	% NODULAR	% LARGE	N	% GOITROUS	N	% NODULAR	% LARGE
0–9	63	25.4	16	0.0	6.2	71	39.4	28	3.6	0.0
10–19	55	72.7	40	2.5	2.5	55	72.7	40	15.0	15.0
20–29	37	62.2	23	26.1	21.7	44	95.5	42	23.8	45.2
30–39	28	67.9	19	47.4	42.1	33	100.0	33	54.6	30.3
40–49	16	75.0	12	66.7	50.0	17	94.1	16	81.2	68.8
50 and over	29	76.9	22	68.2	63.6	28	96.4	27	77.8	81.5
Total	228	57.9	132	29.5	26.5	248	75.0	186	37.1	36.6

these data that if the influence of other etiological factors upon endemic goiter variability is to be studied, sex, age, size, and nodularity should be considered separately. A number of facts reinforce this conclusion: (1) The greater severity of goiter among women, as judged here by greater frequency, larger size, and more nodularity, and also by the earlier appearance of these characteristics, is due to poorly understood mechanisms related to sex. (2) Men frequently remain away from Pedregoso for long periods working in nearby towns or farms, while women and children tend to stay on the reservation. (3) Since most of the goiters become nodular with age, nodularity among younger individuals may have a different meaning from that seen in most older subjects. (4) Estimates of size among older individuals are liable to greater error, because of retrosternal extension, which may have been ignored when diagnosing small goiters, particularly if nodular.

The data should therefore be analyzed both by sex and by age group. In each of these categories, five subgroups have to be studied: without goiter and with small diffuse, large diffuse, small nodular, and large nodular goiters. Since the size of the population and the distribution of goiter in Pedregoso do not generally allow such analysis, more inclusive groups are necessary. In the first decade of life it is only important to compare subjects with and without goiter, ignoring their sex. The second decade is a transitional group, in which large and nodular goiters are still infrequent and cannot be considered apart. Among the adults there are relatively adequate numbers of all groups except that of women without goiter. Finally, among older individuals the meaning of these groups becomes less pre-

TABLE 2. Nodularity and goiter size in Pedregoso

AGE (YEARS)	MEN				WOMEN			
	SMALL GOITERS		LARGE GOITERS		SMALL GOITERS		LARGE GOITERS	
	N	% NODULAR	N	% NODULAR	N	% NODULAR	N	% NODULAR
0–9	15	0.0	1	0.0	28	3.6	0	0.0
10–19	39	2.6	1	0.0	34	14.7	6	16.7
20–29	18	22.2	5	40.0	23	13.0	19	36.8
30–39	11	36.4	8	62.5	23	39.1	10	90.0
40–49	6	50:0	6	83.3	5	80.0	11	81.8
50 and over	8	25.0	14	92.8	5	40.0	22	81.8
Total	97	14.4	35	71.4	118	21.2	68	64.7

cise, except for small diffuse goiters, which are less subject to errors in classification.

Let us first consider the effect of pregnancy upon goiter variability in Pedregoso. In each decade, women with either large or nodular goiters have had a greater average number of pregnancies (Table 3)—which would suggest that gestation increases goiter size and nodularity if this interpretation were not challenged by the fact that the same tendencies are observed among men. Men and women under 30 years of age were not included in Table 3, although they showed these same tendencies. The differences are significant only for men with large as against those with small goiters in the older group ($\text{"t"}_{(31)} = 2.337$; $p < 0.05$). It should be added that men without goiter average a slightly higher number of descendants then goitrous men in these two age groups.

Inbreeding also seems to affect goiter in Pedregoso (Table 4). The prevalence of goiter among children under 10 years whose parents are consanguineous is significantly lower than among children of parents who were born in Pedregoso but in whom consanguinity was not detected ($\chi_{(1)}{}^2{}_{(\text{Yates})} = 4.275$; $p < 0.05$) or children of parents born on different reservations ($\chi_{(1)}{}^2{}_{(\text{Yates})} = 4.275$; $p < 0.05$). The opposite trend seen among persons over 20 years of age is not significant. Inbred adults show a consistent and significant tendency to have fewer nodular goiters ($\chi_{(1)}{}^2{}_{(\text{Yates})} = 6.264$; $p < 0.025$) than those whose parents were born in different reservations. Other comparisons show no significant differences. To sum up, it can be said that inbred people in Pedregoso develop goiters at a later age and that their goiters are somewhat less prone to develop nodules.

PTC-tasting ability was determined, with the Harris and Kalmus technique, in 255 persons in Pedregoso. Since only 4.3 per cent were nontasters, this characteristic could not be properly related to goiter. Nevertheless, a comparison made among tasters showed that both men and women with nodular goiters in the third and fourth decades of life had significantly lower mean threshold values than those with diffuse goiters (*9*).

Height was also correlated with goiter (Table 5), and it was shown that taller subjects of both sexes had larger goiters, in the third and fourth decade. An analysis of variance combining all these groups showed that this relation is significant at the .05 level ($F_{(1,106)} = 4.04$). Nongoitrous men of the same age are in general taller than goitrous ones, but this difference is not significant even when the comparison is made only with males with small goiters ($\text{"t"}_{(50)} = 1.857$).

Nutritional surveys made in Pedregoso revealed two relevant facts: first, the existence of iodine deficiency as judged by an average daily urinary excretion of 33.2 micrograms (*2*); and second, the fact that all year round the people consumed piñon (*1*), which has been demonstrated to be goitrogenic in rats (*42*). Goiter is apparently influenced by these two factors. Subjects with large or nodular goiters have lower mean urinary iodide excretions than those with small or diffuse goiters (Table 6). These differences are better demonstrated by the percentage of subjects in each group excreting 50 micrograms or more. With this approach, statistical significance at the .05 level is reached when large goiters are compared with small ($\chi_{(1)}{}^2{}_{(\text{Yates})} = 4.162$); nongoitrous subjects do not differ significantly from those with diffuse and small goiters. On the other hand, there is a direct relation between mean piñon ingestion and thyroid size (Table 7), nongoitrous subjects

TABLE 3. Fertility and goiter in Pedregoso (total offspring, abortions excluded)

	30–39 YEARS			40 YEARS OR OVER		
GOITER	N	$\bar{X}$	s_e	N	$\bar{X}$	s_e
Women						
Diffuse	11	4.73	0.406	7	7.43	0.922
Nodular	15	5.73	0.547	31	8.29	0.605
Small	18	4.89	0.427	9	7.33	1.414
Large	8	6.25	0.620	29	8.38	0.531
Men						
Diffuse	9	4.88	0.389	11	7.55	0.790
Nodular	6	6.00	0.775	22	9.59	0.973
Small	9	5.00	0.408	13	7.00	1.104
Large	6	5.83	0.792	20	10.20	0.841

TABLE 4. Inbreeding and goiter in Pedregoso (men and women)

		EXAMINED		GOITROUS		
AGE GROUP	INBREEDING	N	% GOITROUS	N	% NODULAR	% LARGE
0–9	a	35	14.3	5	0.0	0
	b	72	38.9	28	3.6	3.6
	c	27	40.8	11	0.0	0
10–19	a	18	61.1	11	18.2	9.1
	b	56	80.4	45	4.4	11.1
	c	35	65.7	23	13.0	4.3
20–29	a	8	87.5	7	0.0	14.3
	b	53	84.9	45	24.4	40.0
	c	19	63.2	12	33.3	33.3
30–39	a	5	100.0	5	40.0	20.0
	b	42	88.1	37	54.0	35.1
	c	14	71.4	10	50.0	40.0
40 and over	a	4	100.0	4	50.0	50.0
	b	43	86.0	37	70.3	73.0
	c	33	87.9	29	82.8	65.5

[a] Parents born in Pedregoso; consanguinity detected.

[b] Parents born in Pedregoso, consanguinity not detected.

[c] Parents born on different reservations.

showing the lowest consumption. Since the values observed differ widely, it is preferable to study in this case the proportion of subjects eating greater amounts, say 100 or more grams daily; again the same trend is seen, the difference being significant when subjects with large thyroids are compared with the rest ($\chi_{(1)}{}^{2}{}_{(\text{Yates})} = 4.061$; $p < 0.025$). Diffuse and nodular goiters do not differ by this approach.

Kinetic studies of iodine metabolism performed in Pedregoso (*3, 4*) are summarized in Figure 1, the most striking findings being (1) comparable and slightly-above-normal figures for net iodine intake by the thyroid and peripheral consumption of thyroid hormones; (2) a considerable "iodine leak," since the total output of iodine by the thyroid is five to ten times the amount incorporated into thyroid hormones; and (3) the abnormal presence in the blood of increased amounts of endogenously labeled triiodothyronine (T_3), iodinated polypeptides (NBEI), and iodotyrosines. These facts, plus increased radioiodine uptake and PBI^{125} levels, express an accelerated turnover of

TABLE 5. Height (centimeters) and endemic goiter in Pedregoso

				GOITROUS											
AGE (YEARS)	NONGOITROUS			DIFFUSE			NODULAR			SMALL			LARGE		
	N	$\overline{X}$	s_e	N	$\overline{X}$	s_e	N	$\overline{X}$	s_e	N	$\overline{X}$	s_e	N	$\overline{X}$	s_e
Men															
20–29	14	162.1	1.85	17	159.8	1.22	6	160.0	2.81	18	159.4	1.41	5	161.5	1.00
30–39	9	164.2	2.41	9	160.8	1.18	8	163.1	1.47	11	160.7	1.02	6	164.2	1.67
40 and over	10	159.5	1.10	11	159.8	1.37	20	159.0	1.48	13	159.0	1.04	18	159.4	1.15
Women															
20–29	2	150.0	2.50	32	150.2	0.90	10	150.5	1.86	23	148.4	1.07	19	152.5	1.01
30–39		—	—	15	150.8	1.54	17	149.0	1.12	22	149.3	1.02	10	151.0	1.07
40 and over	2	150.0	2.50	9	151.9	1.76	30	149.8	0.86	9	150.3	1.88	30	150.3	0.85

TABLE 6. Urinary iodide and goiter in Pedregoso (micrograms per day)

	N	$\overline{x}$	s^2	50 µg OR MORE (%)
Nongoitrous	12	36.6	388.99	25.0
Goitrous				
Diffuse	30	38.1	545.13	26.7
Nodular	37	31.2	324.20	13.5
Small	32	38.5	560.13	31.2
Large	35	30.4	288.24	8.6

TABLE 7. Piñon ingestion and goiter in Pedregoso (grams per day)

	N	$\overline{x}$	s^2	100 G OR MORE (%)
Nongoitrous	22	46.0	1604.67	4.5
Goitrous				
Diffuse	43	61.6	3315.72	18.6
Nodular	50	75.7	13665.77	16.0
Small	41	57.5	2921.71	9.8
Large	52	78.4	13462.44	23.1

radioiodine by the gland and considerable inefficiency in handling iodine in terms of the percentage used for the synthesis of thyroid hormones. Some of these findings have been found in other instances of endemic goiter in America (*31, 41*) and elsewhere (*12*).

How should radioiodine studies be correlated to goiter variation? The PBI^{125} reflects the speed of the thyroid in incorporating radioiodine into circulating iodinated compounds, and Table 8 shows that nodular goiters have significantly higher values for this parameter, as is also true for NBEI^{125}. In a previous publication (*4*) we have shown that nodular goiters have the widest range of values of thyroidal iodine pool, comprising both the smallest and the largest figures observed. This finding may explain why PBI^{125} values have the greatest variance among nodular goiter, small pools determining the high values observed. In support of this view, the thyroidal

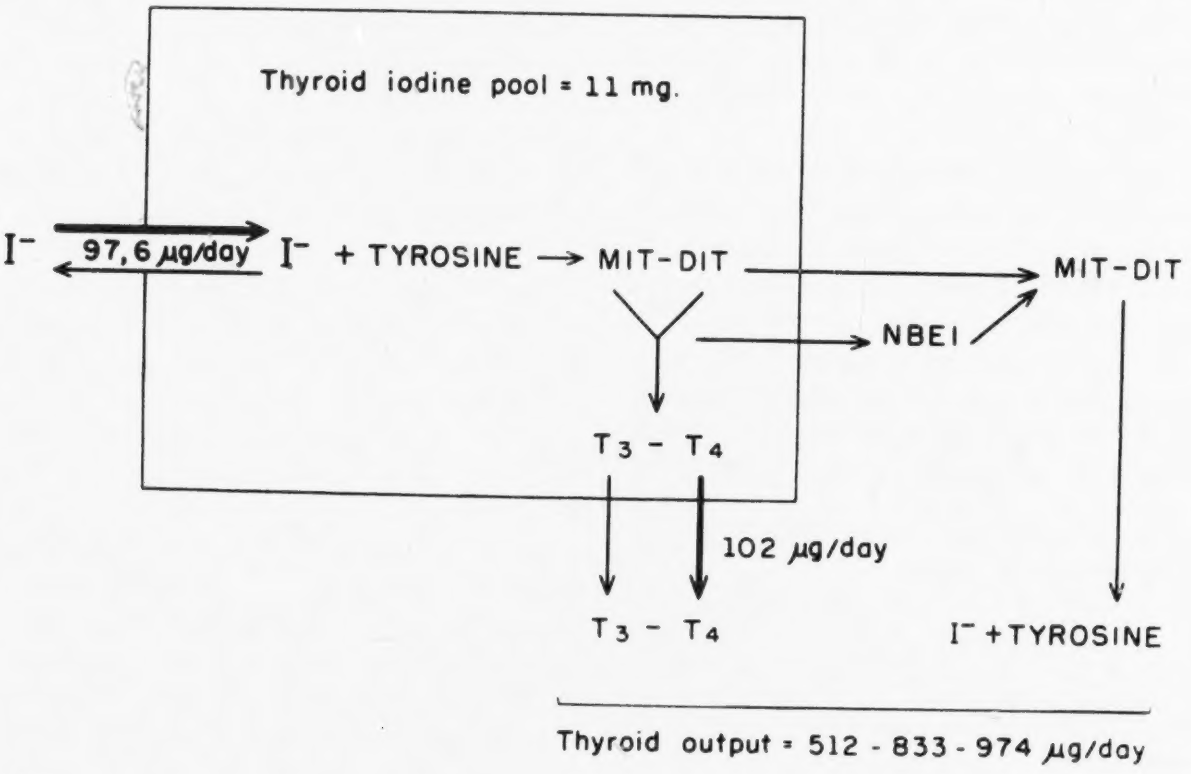

FIGURE 1. Iodine metabolism in Pedregoso

TABLE 8. PBI^{125} and $NBEI^{125}$ in Pedregoso (%/dose/liter)

	PBI 125			NBEI 125		
	N	$\bar{x}$	s^2	N	$\bar{x}$	s^2
Nongoitrous	10	0.17	0.0135	7	0.012	0.0^4176
Goitrous						
Diffuse	30	0.18	0.0204	19	0.012	0.0^4823
Nodular	34	0.59	0.1874	29	0.028	0.0^38736
Small	30	0.31	0.1147	23	0.022	0.0^37575
Large	34	0.39	0.1529	25	0.022	0.0^35058
F for nodularity		13.709 [a]			8.005 [b]	
F for size		2.323			0.097	

[a] $p < 0.001$.
[b] $p < 0.010$.

iodine pool among 21 subjects in Pedregoso, calculated by the Riggs isotopic equilibrium method (*4*), shows a significant inverse relationship with the PBI^{125} values observed in these subjects ($r = 0.472$; $p < 0.025$). A similar correlation has been observed in euthyroid patients in Scotland (*45*). Furthermore, small iodine pools could also explain high $NBEI^{125}$ values, since this parameter shows a significant positive correlation with PBI^{125} ($r = 0.50$; $p < 0.001$).

Endogenously labeled triiodothyronine is chromatographically present in the same proportion among all goitrous subjects and absent in the nongoitrous (Table 9), although the difference is not significant ($\chi_{(1)}{}^{2}{}_{(\mathrm{Yates})} = 2.549$). On the other hand, endogenously labeled iodotyrosines were present in a significantly higher percentage of the Indians with nodular goiters than of those with diffuse goiters ($\chi_{1(\ )}{}^{2}{}_{(\mathrm{Yates})} = 5.711$; $p < 0.01$), while there is no significant difference between subjects with small and large goiters. Nongoitrous persons have labeled iodotyrosines in a smaller proportion than goitrous ones, particularly in comparison to those with nodular goiters. Subjects with labeled iodotyrosines have significantly higher PBI^{125} values ("t"$_{(46)}$ = 2.31; $p < 0.05$); this is not true for those with labeled triiodothyronine. The demonstration of iodotyrosines can be directly ascribed to small thyroid iodine pools, as is suggested by the fact that six subjects in whom they were present had an average pool of 1.97 mg ± 0.327, while five others in whom they were not demonstrated had an average value of 6.22 mg ± 1.757 ("t"$_{(4)}$ = 2.37; $p < 0.05$).

TABLE 9. Endogenously labeled triiodothyronine and iodotyrosines in Pedregoso

	T_3		IODOTYROSINES	
	NO.	%	NO.	%
Nongoitrous	8	0.0	14	21.4
Goitrous				
Diffuse	11	36.4	28	10.7
Nodular	25	36.0	34	41.2
Small	14	35.7	26	19.2
Large	22	36.4	36	33.3

Discussion

Multiple causes are involved in the etiology and pathogenesis of endemic goiter, many of which still require proper identification and evaluation. Iodine deficiency is usually the predominant etiological factor; other environmental influences are probably also present and, where there is adequate iodine, would sometimes suffice to explain endemic goiter. Natural goitrogens are the best-known example. Genetic factors have not been shown to influence the prevalence of endemic goiter, although the possibility of their playing some role cannot be discarded.

The participation of all these factors should be studied in relation not only to the occurrence but also to the variability of goiter. Such studies are seldom made, presumably because of the difficulties in definition and methodology that have been mentioned. It is upon variation that genetic factors have been demonstrated, as exemplified by the association of PTC-tasting ability with nodular goiter. Weak environmental etiological factors may also be better studied with this approach, when a predominant cause like iodine deficiency is present.

In Pedregoso we have looked into goiter variation with three different elements in mind: presence, size, and nodularity of goiter. These obviously interact in some measure, but each is characterized in our data by a particular constellation of statistically significant factors. Nodularity is related to the two clearly defined genetic aspects studied so far—inbreeding and PTC-tasting ability—and to some radioiodine parameters. Inbred people develop fewer nodular goiters; Indians with nodular goiters have lower thresholds for tasting PTC; and radioiodine studies show high PBI^{125} and NBEI^{125} values and a greater proportion of endogenously labeled iodotyrosines in subjects with nodular goiters. All these radioiodine findings can be linked to the predominance of small thyroidal iodine pools among those with nodular goiters. Large goiters, on the other hand, are associated with the two best-defined environmental factors studied by us: the subjects eat less iodine and more piñon. Size is also positively associated with two complex factors—height and fertility. Presence, unlike the other two elements, has been connected significantly only with inbreeding and during the first decade. It is noticeable here that early appearance of goiter, like nodularity, is negatively influenced by inbreeding. Sex and age, as such, are involved with all three elements, but we feel that their relevance should be sought mainly through interaction types of effects.

In view of the complexity and number of factors to be considered acting together, it would seem necessary to apply to all these data a multivariate analysis through which the proper value could be attached both to main factors and to the many kinds of interactions present. This might not be possible, however, for two main reasons: the small size of our sample, especially since the subsamples in which parameters were recorded did not always comprise the same individuals, and the fact that the categories of goiter contain different types of error in a nonrandom way and have different meanings. For example, relationships with age are difficult to study because small diffuse goiters at an early age may develop into large and/or nodular goiters. Our interpretation of the Pedregoso data requires us to extend these studies to other similar Pewenche communities, but even then comparisons with entirely different situations using similar types of analysis would be necessary to determine the bearing of such findings on the general problem of endemic goiter. Is endemic goiter the adaptation of man to iodine deficiency? Our data lead us to suggest that subjects with certain types of goiter are better adapted than others. The fact that Indians with large goiters are taller and have more children might be in line with such an interpretation, even if we are unable to interpret the mechanisms involved.

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DISCUSSION

J. D. Niswander

The speakers today have each considered one special disease problem of the Indian. I should like to discuss three of these in relation to one another. I will discuss diabetes mellitus, gallbladder disease, and nutrition in the context of the over-all variability in frequency of diabetes among various Indian tribes in the United States.

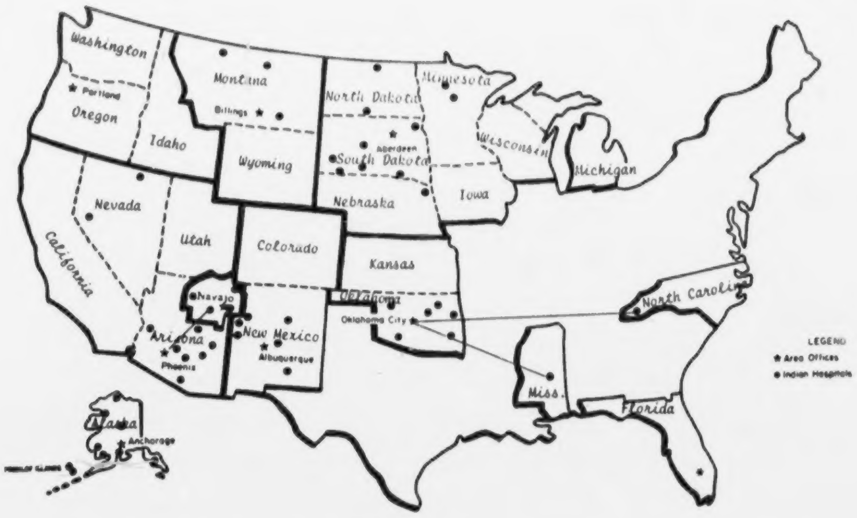
Specific studies have reported an unusually high frequency of diabetes among several North American tribes—with the studies being conducted among the Pima, reported by Dr. Miller, being the foremost example. There are, however, very few data on the question of just how prevalent diabetes is throughout the various American Indian groups or whether it is more or less uniquely confined to particular tribes. Certainly such a hypothesis as that put forward by Dr. Neel—that diabetes may represent the effects of changing dietary patterns on genomes previously adapted to a "feast or famine" situation—would suggest that a high frequency of diabetes might be expected among populations who have been both recently and rapidly transformed from this primitive pattern to a more sedentary and overfed condition.

The only source of information that I am aware of on this subject, although the data are less than ideal, is the records of the U.S. Public Health Service, Division of Indian Health Hospitals. Accordingly, I have obtained tabulations of discharge diagnoses from these hospitals for the four-year period 1963–1967. This represents approximately 250,000 discharges. I have tabulated the proportion of discharges from each hospital in which the primary diagnosis was diabetes mellitus. The rank order for the hospitals changes very little if, instead of the total number of discharges, the total beneficiary population of each hospital is used as the denominator. I believe there is enough uniformity between Public Health Service Indian Hospitals to make these data reasonably adequate for relative frequency comparisons, although one certainly would not want to attribute a great deal of precision to them.

The map shows the location of the various Public Health Service Hospitals throughout the United States and their grouping into seven administrative areas. The eighth area (Portland) delivers service through contract with local facilities and not directly through Public Health Service Hospitals.

The mean frequency for the 46 Indian hospitals is 2.2 per cent. This compares with figures of 1 to 1.5 per cent for similar data for several surveys of general non-Indian hospitals throughout the United States representing over 26,000,000 hospital admissions.

The table groups the hospitals by the relative frequency of admission for diabetes and indicates the major tribes served by each. As can be seen, the range expressed by the mean frequency of 2.2 per cent is very wide, with roughly a twentyfold difference between the lowest and the highest. The table also shows the relative frequency of admission for gallbladder disease, which very closely parallels that for diabetes. In fact, the rank correlation is slightly higher than .5 and extremely significant. There is, however, a considerably narrower range of variability than


Washington
Portland
Oregon
Idaho
Montana
Billings
Wyoming
North Dakota
Minnesota
Aberdeen
South Dakota
Wisconsin
Michigan
Nebraska
Iowa
Nevada
Utah
Colorado
California
Kansas
Oklahoma
Oklahoma City
North Carolina
Navajo
Arizona
Phoenix
New Mexico
Albuquerque
Miss.
Florida
Alaska
Anchorage
LEGEND
★ Area Offices
● Indian Hospitals

* Seminole Indian Reservation Services Administered From the Oklahoma Indian Health Area Office.

for diabetes. It can be seen that there is an equally striking association between diabetes and average birth weight. Here again the regression of birth weight on frequency of diabetes, by hospital, is highly significant.

Specifically in regard to diabetes there are several points of interest. First of all, in almost two thirds of the Indian hospitals diabetes seems to represent a significantly greater proportion of the patient population than in non-Indian hospitals. Most of the major linguistic groups are represented in the high and intermediate diabetes categories with the exception of the Aleut-Eskimo group and possibly the Athabascans. The highest diabetes group is composed of several different tribes, but most conspicuous are the so-called five civilized tribes from the southeastern portion of the country. All had relatively highly developed agriculture prior to European contact. Also notable among this group are several of the southwestern agricultural tribes including the Pima. It might be suspected that these early agriculturists would be the ones most susceptible to acculturation and hence would have undergone the most significant changes in dietary patterns.

The low-diabetes group is notable for including all of Alaska—the Aleuts, Eskimos, and various Alaskan Indians—and also the Athabascan-speaking Navajo and the closely related Apaches. I think it is noteworthy that the Alaskan natives and the Navajo are probably the least acculturated of all the Indians remaining in the United States.

The possibility that their low frequency of diabetes is due to the inherent genetic make-up of these groups, independent of environmental factors, cannot be excluded. Likewise, it could be argued that birth weight and diabetes are related by virtue of both being the result of a particular genetic constitution. Alternatively, the birth-weight gradient might be considered a secondary effect of maternal diabetes. It seems to me, however, that the relationship between gallbladder disease, birth weight, and diabetes, when viewed together with the admittedly limited information on cultural variation, suggests at least one other possibility: the operation of common environmental factors, presumably dietary. To speculate a bit more than is perhaps warranted by the data, I would suggest that virtually all Indians may be of a genetic constitution especially susceptible to diabetes and its expression is the result of dietary and nutritional factors associated with present-day reservation life.

In this regard, it is unfortunate that there are

Frequency of diabetes mellitus and gallbladder disease and mean birth weight in Public Health Service, Division of Indian Health Hospitals

HOSPITALS	MAJOR TRIBES	DIABETES * (% OF ALL ADMISSIONS)	GALLBLADDER * (% OF ALL ADMISSIONS	MEAN BIRTH WEIGHT †
Window Rock Area (6 hospitals—New Mexico, Arizona)	Navajo	0–1	1.9	6.68
Belcourt, North Dakota	Chippewa			
Mescalero, New Mexico; White River, Arizona	Apache			
Keams Canyon, Arizona	Navajo, Hopi			
Alaska (7 hospitals)	Aleut, Eskimos "Alaskan Indians"			
Montana (3 hospitals)	Blackfoot, Crow Cheyenne, Gros Ventres Assiniboine, Cree	2–3	2.3	6.88
Cass Lake, Minnesota; Red Lake, Minnesota	Chippewa			
North Dakota, South Dakota, Nebraska (6 hospitals)	"Sioux"			
Santa Fe, New Mexico; Zuni, New Mexico	"Pueblo," Zuni			
San Carlos, Arizona	Apache			
Clinton, Oklahoma; Lawton, Oklahoma	Cheyenne, Arapaho, Kiowa, Comanche, Caddo			
Sells, Arizona	Papago			
Cherokee, North Carolina; Tahlequah, Oklahoma	Cherokee	4–10	2.6	7.05
Claremore, Oklahoma; Pawnee, Oklahoma; Philadelphia, Mississippi	Cherokee, Creek, Ponca, Choctaw, Seminole			
Talihina, Oklahoma	Choctaw, Chickasaw, Cherokee, Creek			
Wagner, South Dakota	Sioux			
Sacaton, Arizona	Pima, Papago			
Shurz, Nevada; Owyhee, Nevada	Paiute, Shoshone			
Winterhaven, California; Parker, Arizona	Cocopa, Mohave, Chemehuevi			

* Based on 257,320 discharge diagnoses, 1963–1967.
† Based on 27,288 births.

no very adequate data available on relative obesity between tribes or on the increase in obesity that may have occurred in recent years. There is, however, one study that reports approximately a six-kilogram increase in weight among the Navajo, with no increase in stature, over a twenty-year period. The reservation Navajo, however, are still considered relatively lean in body build, without the great tendency toward obesity that is seen in some of the other tribes. It would be of considerable interest to follow Navajos who have now moved to the major urban centers and compare them both for obesity and for serum glucose levels with their presumably less acculturated counterparts who have remained on reservations.

My purpose in this discussion has been primarily to point up the need not only for detailed studies of special diseases in specific groups but also for data giving comprehensive coverage over many Indian populations. Certainly, studies of human adaptation and attempts to understand the interactions between genotype and changing cultural patterns demand considerable knowledge of the variation which may exist within and between various human populations.

If my speculations prove to be correct, diabetes may become an increasingly important medical problem as the acculturation of the American Indian increases. Further, such considerations would seem important in terms of general planning for dealing with the health problems of emerging primitive groups.

Acknowledgment

I am grateful to Mr. Abraham Ranofsky, Program Analysis and Statistics Branch, Division of Indian Health, for furnishing the special tabulations on diabetes and gallbladder disease.

Edmundo Covarrubias

The presentations we have heard this afternoon make it clear that the American Indians received from the conquisatadors a rather heavy burden of evils: biological agents of disease and the pressure of a different culture, stresses to which they had to become adapted at the toll of increased mortality and morbidity figures. Difficult related questions, not yet answered, concern the proportion of genes potentially capable of provoking disease that have passed to the Indians from the other races with which they are in the process of mixing.

All the specific health problems mentioned in this session are characterized by a complex causality involving multiple genetic, environmental, and cultural factors. The genetics of a disease like diabetes is a complicated subject, in which no precision as to the part played by genes has thus far been reached; moreover, diabetes may represent a genetically heterogeneous group of disorders.[1] A similar situation exists for endemic goiter. In the case of malaria, in which increased resistence is found among individuals heterozygous for hemoglobin S, other still unknown genetic factors, probably polygenic, may be involved.[2]

What I have said in relation to genetic etiology is paralleled in the field of environmental causes. Thus, the variability of all these diseases is caused by an interaction of factors that is probably unique in each population, and no geneticism or environmentalism will be of real value to an over-all comprehension of the problems.[3]

Among the specific questions raised to-day, I should like to point out the associations between gene distributions and such phenomena as the relationship suggested for Pewenche Indians between nodular goiter and lower phenylthiocarbamide thresholds in the group of "tasters" of this substance. Similar results may appear from the studies of diabetes and gallbladder disease among the Pima Indians, in relation to blood groups. Whether or not such associations are real is to be found in the future, but they may constitute starting points for the analysis of polygenic systems in man. Another interesting question here concerns how the epidemiology of a disease would be influenced by the distribution of genes, which vary notably from tribe to tribe, as has been shown for blood groups in some of the papers submitted today.

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The mental diseases present a set of problems even more formidable in view of the difficulties of diagnosis due to cultural differences. One effort in this direction was recently made in Chile: a new structural theory of psychoses led to transcultural definitions that made it possible to distinguish considerable differences in the frequencies of the proposed types of psychoses between Mapuche Indian patients and the controls.[4]

Finally, I feel tempted to generalize to other systems the words of Böök[5] concerning the etiology of mental illness: ". . . my general medical and biological background together with my fragmentary knowledge of psychiatric research work has led to the tentative conclusion that any unitary explanation of the etiology even of what is now considered a special type of mental illness, or a clinical entity, will not contribute to the advancement of psychiatric research."

To sum up, biomedical research among the American Indians is apt to disclose new facts that may be of value both for specific public health policies and for a general understanding of the diseases studied.

General Discussion

Jelliffe: I should like to congratulate Dr. Béhar on his excellent paper. It seems to me that, late as the hour is, it would be wrong to let this go by without emphasizing its importance. As those of us here who are working in the field of childhood malnutrition will agree, the lessons to be learned from his paper are of great importance.

First, I think that we all have to realize that all groups that have been living in a certain ecosystem for many centuries have evolved and adapted their own practice of infant feeding, and that if we rush in with our own outside ideas without taking these into careful consideration we may do very great harm.

Second, there are lessons to be learned, I believe, from different groups of people and different traditional systems. I think that we in the so-called Western world have a considerable body of dogma in our own infant feeding that in fact might very well be re-examined in the light of practice elsewhere. For example, as Dr. Béhar pointed out, in most of pre-conquistador South America and also in Polynesia animal milk was not traditionally drunk. Yet people there reared children and fed them perfectly adequately without it. There are messages for us from these different groups.

Another important aspect of Dr. Behar's paper is the fact that disruption of a culture can very frequently have its most sensitive and devastating effect on the age group that is most precariously poised physiologically. That, of course, is the young child—the weanling, who is in a process, if you like, of biocultural transition.

When we consider the Indian communities in the Americas, we should, I think, bear in mind what has happened elsewhere in the world. For example, the Hadza hunters of northern Tanzania were surveyed some six years ago and were found to have a pattern of infant feeding well adapted to their community—breast feeding, bone marrow, pre-chewed meat, and so forth. Five years later another survey was carried out, and the pattern of infant feeding and childhood malnutrition had changed completely. In the first survey there was very little protein-calorie malnutrition. In the second it was very common.

What practical messages are there to be gained from Dr. Béhar's paper? I think there are these: We should realize that as well as the rapid spread of infections due to viruses, such as measles, or to bacteria, such as tuberculosis, the spread of inappropriate non-adapted ideas concerning infant feeding can have a very considerable effect on local child health and on the local pattern of childhood malnutrition. How can we try to prevent this? I think that very probably, before changes are allowed to occur too easily in the dietary habits of American Indian groups, careful thought should be given to who has been responsible for these changes in the past.

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Many of these inappropriate imported changes are iatrogenic, using the term in its wide sense. I think, therefore, that in dealing with the American Indian groups that have been discussed here the infant feeding practices taught by the health services should be most carefully adapted to the local circumstances and should not merely be cultural imports from elsewhere.

Furthermore, any imports of foods into these communities through, for instance, food aid programs undertaken by international agencies, national services, and voluntary organizations, including missions, should be very closely looked at for whether their hoped-for immediate benefits will in fact yield ill effects in the near future.

Lastly, "commerciogenic" malnutrition has to be mentioned here. It is probably as dangerous to introduce a commercial milk company, a company selling milk and promoting feeding bottles, to a community of people who are successfully breast-feeding as it is to introduce measles or tuberculosis.

Waterlow: May I ask Dr. Giglioli one question? Is it your belief that malaria did not originally exist in the Amazon forest country? Was it one of the things brought in along with others?

Giglioli: My paper touches on this question. Not that I have any competence to discuss it. I mean, it is a moot question whether malaria pre-dated the Conquest or was introduced. I think the consensus now is that it was introduced and that this is probably the reason why the Amerindian reacts so much more actively to malaria infection than the Negro, who has been exposed to it for ages.

We have no data to go by. Consider the Mediterranean populations, which were exposed to malaria for centuries. Certainly there was no such total endemic malaria. People suffered from malaria and died from it and never came to terms with it as the African does. You do find endemic malaria in a few other groups—in northeastern India, for example. But it is so localized as to suggest a racial factor in the mechanism of holoendemic malaria quite apart from length of exposure.

Waterlow: That is what I am getting at—whether there is a racial factor even with the Amerindian.

Giglioli: I think so.

Roche: What has been said about care with the food brought into these areas induces me to say a few words about the moral issues involved in studying Indians. These are people who are at our mercy both morally and physically. The Declaration of Helsinki on research on human beings states that the *informed assent* of the people on whom one is doing research should be obtained. This includes research on their internal milieu, not merely their external milieu. The persons responsible for the statement were not, I believe, thinking about this type of study because it is practically impossible, as everybody will agree, to get really informed assent from the Indians. This means that the burden is on the investigator, who must exert the utmost restraint in his investigation to be sure that his activity is in no way harmful to the populations he is studying.

Cohen: I want to return to the theme of how these factors might emerge without a basic genetic alteration. I raise the question whether we are not dealing with the well-known modulation effects that derepressors and repressors might have on the transcription and translation phenomena. It is now, I think, reasonably well documented that the influence of a variety of hormones can lead to the expression of enzyme activity that is not seen at a lower level of certain hormones.

As a matter of fact, many hormonal patterns are now being explained in molecular biological terms of messenger RNA and all that goes with it. This must mean that there is an interplay by a variety of factors on the genetic potential of an individual to give different degrees of quantitative expression. I am not sure there would be qualitative changes.

As I listened to many of these presentations, diabetes in Indians and so on, I thought it quite conceivable that this could be a quantitative variation of one or another enzyme pattern that would lead to a metabolic anomaly of the kind I have referred to. Considering the influence of diet on a number of these circumstances, I think the question still remains whether the basic genetic pattern is being significantly altered in the traditional sense or whether the change is taking place at the modulating level, as it were.

Moderator: A discussion of this would take us rather far afield. I am not sure whether you are using the term *modulation* in the sense used by the bacterial geneticist or whether you are referring to the kind of nature-nurture interactions for which we have come to have great respect in man. The idea that in fact the genetic readout might be different for ourselves than for, say, the Indians simply because of the environmental difference is a challenging idea. My first reaction is that the homeostatic mechanisms of man are such as to buffer this readout quite a bit. On the other hand, perhaps we should devote some thought to trying to put that idea to some kind of critical test, and certainly one opportunity is in transitional populations. For instance, if it can be documented that the polymorphisms, which are a readout for proteins, alter in frequency more rapidly in a population in cultural transition than can possibly be ascribed to genetic selection, this would be hard evidence that the readout is different. That is the kind of evidence that I think it would take to satisfy most of us.

Cohen: Not to prolong this, it is very clear that a variety of nutritional states will elicit a great pattern of enzymatic changes that are not due to an instantaneous transition of the genes. We know this. Just a simple high carbohydrate intake as against a low, high nitrogen intake as against low, will cause changes of level of high orders of magnitude. These can be feedbacks of various kinds, but many have hormonal factors that are known to have their initiating effect on the transcription phase in which the gene may be repressed for a particular level of quantitative operation.

I am only suggesting that before we get too involved in genetic markers as an absolute known relationship, which is clearly the case in many diseases that have been defined in these terms, we give some thought to the possibility that in many of these adaptive phenomena we are dealing with something quite different.

I hope that at some time in the future there may be a chance to talk at this level as a follow-up to our discussion today. This is where the new molecular biology may perhaps change some of our concepts.

Moderator: The final item on the agenda is labeled "Commentary and Summarization." This will be extremely brief. One of the marks of a good meeting is the extent to which old ideas are rescrutinized and questioned, and from beginning to end of this session we have been questioning some of our old ideas, beginning with the matter of the origins of the American Indians and ending with details of disease susceptibility and resistance. The session has been very profitable, and it is my privilege to thank PAHO for making this gathering possible, both financially and in terms of facilities.

Appendix

TABULATIONS OF PHENOTYPE AND GENE FREQUENCIES FOR 11 DIFFERENT GENETIC SYSTEMS STUDIED IN THE AMERICAN INDIAN[1]

R. H. Post, J. V. Neel, and W. J. Schull

With the impetus given to further genetic studies of the American Indian by the forthcoming International Biological Program, it is probable that the next decade will witness a sharp increase in investigations of this kind. The literature is already voluminous and scattered; it seemed that a compilation of those studies which deal with the major genetic polymorphisms would prove useful in assisting investigators both in planning and facilitating studies and in comparing their findings with those of others. Accordingly, in Tables 1 to 11 tabulations are presented for the following systems: ABO, MN, Rh, Kidd, Duffy, Diego, P, Lewis, transferrins, haptoglobins, and Gm. These are all systems exhibiting marked polymorphism. We have not included systems, such as the glucose-6-phosphate dehydrogenase or hemoglobin system, that are polymorphic in some populations but essentially nonvariant in the Indian. Nor have we included some systems that while probably polymorphic are still inadequately defined either genetically or phenotypically, such as sensitivity of taste to phenylthiocarbamide, distribution of hair on phalanges, tongue-rolling, and so on. An effort has been made to survey all publications prior to November 1967; these are cited in the Bibliography.

In preparing the tabulations, a recurrent question in the face of an unusual finding has been whether the cause might be racial admixture (i.e., miscegenation). Furthermore, as we have pointed out elsewhere (*5*), the Indian presents an unusual challenge to those interested in recent developments in genetic taxonomy (see *1, 2*), but attempts to apply these developments may be vitiated by substantial racial admixture. Accordingly, we have made an effort here to classify each sample in one of three ways: (1) probably essentially no admixture, (2) non-Indian contribution to the gene pool probably less than 5 per cent, and (3) non-Indian contribution to the gene pool probably greater than 5 per cent.

As every student of the problem knows, the classification of certain samples into one of these three categories is difficult, and we are sure not only that there are grounds for issue with some of our decisions, but also that some of these decisions are incorrect and will have to be modified. In this connection, it should be emphasized that the decision applies only to the particular sample that was examined, and not necessarily to the entire group or tribe. Each classification has been based on the history of the tribe, the opinion of competent observers, the results of the genetic studies themselves, and, where given, the author's statement.

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[1] Work supported in part by U.S. Atomic Energy Commission Contract AT(11-1)-1952.

The results of typings for the ABO system have been thought to provide especially critical material for decisions regarding admixture. We accept the working hypothesis that I^B and I^{A_2} are not found among American Indians and that I^{A_1} does not occur in South America. However, we note that in the few South American samples where I^B and I^{A_1} do occur, the ratio of the frequency of the former to that of the latter tends to be higher than in western Europe and Africa, the presumed origin of these genes. The possibility must be recognized that I^B was present in South America in pre-Colombian times (see also *6*), perhaps introduced by trans-Pacific contacts.

Additional genetic grounds for decisions regarding sample purity are the presence of the following antigens, wherever tested for: Kell, Lewis (a), Berrian (Be), Henshaw (He), Lutheran (Lu^a), Miltenberger (Mi^a), Sutter (Js^a), V of the Rh system, Verweyst (V^w) of the MN system, and Wright (Wr). All of these are thought to be absent in unmixed Indians, and we have not included tabulations for any of them. References establishing the virtual or complete absence of these traits in samples of unmixed Indians are listed in Table 12. We have felt, on the other hand, and contrary to some opinions, that the occurrence of Rh-negative individuals (d) need not necessarily indicate admixture (see *3*), although high frequencies may be of significance.

In our opinion, many types of critical comparisons are impossible when there is more than 5 per cent admixture in the sample. Only the data on samples with no admixture, or less than 5 per cent admixture, have been incorporated into Tables 1 to 11, with samples of the first class designated by asterisks. The references to samples with more than 5 per cent admixture are given in Table 13, which lists the samples in alphabetic order.

The names of tribes and groups are those given by authors, to which an arabic numeral has been added in cases of two or more samples of the same name. Among the civilized populations of Middle and South America, where tribal organization has long since disappeared, these names may be confusing and occasionally meaningless. Thus there are two samples of Mayans so called by the authors, an unsatisfactory name since several other samples are tabulated of Mayan-speaking Indians—Chol, Kekchi, Mam, Pocomam, Quiché, and others. Five samples are designated Aymara, two Quechua, and one Chibchan, each of which is a large linguistic stock including several distinct cultures and several millions of persons. Three linguistic stocks of simpler culture are similarly employed as names of samples—Arawak, Carib, and Guaraní. From four to ten samples are designated by tribal names that fall under each of these stocks.

The estimates of gene frequencies in the tables that follow were computed on an IBM 1130 Model, using programs written in the Department of Human Genetics. The ABO program has been published (*4*); the other programs are available on request. The "goodness of fit" of the observed phenotype frequencies with those expected under Hardy-Weinberg equilibrium and with the calculated gene frequencies has been tested by chi square. The latter are not given, but the corresponding probabilities when significant are indicated by one of three letters in the column to the far left of each table, as follows: A $- 0.05 > P > 0.01$; B $- 0.01 > P > 0.001$; C $- 0.001 > P$. The validity of applying such χ^2 tests to small samples in which the mean coefficient of relationship is probably not negligible is dubious, but we have followed convention in this respect.

Names of countries are abbreviated by three letters and followed by a brief indication of locality, such as that of a state or province, River (R), Mountain (M), Lake (L), or region (N for north, and so on). Exceptions are made for French Guiana and for Guyana and Surinam (formerly British and Dutch Guiana, respectively), for which no locality is added, since their areas are small.

In several instances an author has published data on two or more "subtribes" as well as the pooled data of the "tribe." The grounds for recognizing subtribes are seldom clear; they sometimes appear to represent different villages of the same tribe. In all cases, we have retained the author's designation but listed his data under the main tribal unit, with a cross-reference under subtribe. Thus, the data on the Arecuna subtribe of the Pemons will be found under "Pemon-Arecuna" with a cross-reference under "Arecuna."

Genetic marker data have been published on a few samples of mixtures of several tribes or groups that appear to have less than 5 per cent non-Indian ancestry. Such data are not tabulated here, but the references are listed apart (Table 14) in alphabetical order of the tribes, together with notes on geographic locality, the names of the several tribes included in the mixture, and our estimate of the amount of non-Indian ancestry of the pooled sample.

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CODE

General

1. An asterisk following the name of a tribe or group indicates essentially no Negro or Caucasian admixture.

2. When two or more samples are presented from a tribe or group of the same name (whether from the same or different authors), each sample is arbitrarily assigned a serial number. This appears in column 14 unless the sample is pooled from several areas or subtribes, in which case "P" appears in column 14 preceded by the serial number in column 13. (Subsamples are presented separately, also, whenever available.) "P" stands for "pooled."

Abbreviations of nations and colonies and other abbreviations

Nations

Argentina	ARG
Bolivia	BOL
Brazil	BRZ
Canada	CAN
Chile	CHL
Colombia	COL
Costa Rica	COS
Ecuador	ECD
Guatemala	GUA
Honduras	HON
Nicaragua	NIC
Panama	PAN
Paraguay	PAR
Peru	PRU
Salvador	SAL
United States	USA
Venezuela	VEN

Colonies

British Honduras	BRH
French Guiana	FRG
Guyana	GUY
Surinam	SUR

Other abbreviations

North	N
East	E
South; Saint, Sierra	S
West	W
Mountain(s)	M(TS)
Another; Others	ANO
River	R
Lake	L
Delta	D
Island(s)	I(S)
Pooled data, from several samples	P

TABLE 1A. THE A1, A2, B, AND O GENE FREQUENCIES, AS DETERMINED WITH ANTI-A, ANTI-A1, AND ANTI-B SERA.

TRIBE OR GROUP	NATION	REGION	REF	SAMPLE SIZE	PHENOTYPES						GENE FREQUENCIES				P
					O	A1	A2	B	A1B	A2B	A1	A2	B	O	
ACAWAIO*	GUY		130	90	90	0	0	0	0	0	0.000	0.000	0.000	1.000	
AGUARUNA*	PRU	R MARANON	168	151	151	0	0	0	0	0	0.000	0.000	0.000	1.000	
ALACALUF 3	CHL	MAGELLAN	164	44	41	3	0	0	0	0	0.035	0.000	0.000	0.965	
APACHE* 1	USA	ARIZ	042	179	105	74	0	0	0	0	0.234	0.000	0.000	0.766	
APACHE 2	USA	NEW MEX	090	108	55	48	1	2	2	0	0.266	0.006	0.019	0.708	
ARAWAK*	SUR		060	88	87	0	0	1	0	0	0.000	0.000	0.006	0.994	
ARECUNA	SEE	PEMON													
ATACAMENOS 1	CHL	ANTOFAGST	164	80	73	3	0	4	0	0	0.019	0.000	0.025	0.955	
ATHARASCAN 1	USA	ALASKA	062	78	74	4	0	0	0	0	0.026	0.000	0.000	0.974	
AWEIKOMA ANO*	BRZ	S CATRINA	233	122	121	0	0	1	0	0	0.000	0.000	0.004	0.996	
AYMARA* 1	PRU	PUNO	028	58	58	0	0	0	0	0	0.000	0.000	0.000	1.000	
AYMARA* 3	PRU	PUNO	168	93	93	0	0	0	0	0	0.000	0.000	0.000	1.000	
AYMARA* 4	BOL	3 AREAS P	169	503	491	7	3	2	0	0	0.007	0.003	0.002	0.987	
BARI*	VEN	W ZULIA	132	126	126	0	0	0	0	0	0.000	0.000	0.000	1.000	
BRIBRI*	COS	SALITRE	177	50	50	0	0	0	0	0	0.000	0.000	0.000	1.000	
CARECAR*	COS	UJARRAS	177	62	62	0	0	0	0	0	0.000	0.000	0.000	1.000	
CAINGANG	BRZ	R G SUL	231	341	334	4	1	2	0	0	0.006	0.001	0.003	0.989	
CAKCHIQUEL 1	GUA	L ALTIILN	171	150	142	4	0	4	0	0	0.013	0.000	0.013	0.973	
CAKCHIQUEL* 2	GUA	SOLOLA	170	9	9	0	0	0	0	0	0.000	0.000	0.000	1.000	
CAKCHIQUEL 3	GUA	SUMPANGO	268	137	132	2	0	3	0	0	0.007	0.000	0.011	0.981	
CAMARACOTO	SEE	PEMON													
CAMPA*	PRU	R URUBAMB	168	89	89	0	0	0	0	0	0.000	0.000	0.000	1.000	
CAYAPA* 2	ECD	R CAYAPAS	167	244	240	0	0	4	0	0	0.000	0.000	0.008	0.992	
CHACOBO*	BOL	R YATA	169	14	14	0	0	0	0	0	0.000	0.000	0.000	1.000	
CHAMA*	BOL	R MADIDI	169	30	30	0	0	0	0	0	0.000	0.000	0.000	1.000	
CHAMULA*	MEX	CHIAPAS	024	63	63	0	0	0	0	0	0.000	0.000	0.000	1.000	
CHEROKEE*	USA	N CARLINA	209	78	75	0	1	2	0	0	0.000	0.006	0.013	0.980	
CHOCO*	PAN	DARIEN	178	80	80	0	0	0	0	0	0.000	0.000	0.000	1.000	
COLORADO* 2	ECD	S DOMINGO	167	36	36	0	0	0	0	0	0.000	0.000	0.000	1.000	
CUIVA*	VEN	APURE	134	82	82	0	0	0	0	0	0.000	0.000	0.000	1.000	
CUNA* 2	PAN	S BLAS IS	178	388	388	0	0	0	0	0	0.000	0.000	0.000	1.000	
DIEGUENO	USA	CAL	202	58	56	1	0	1	0	0	0.009	0.000	0.009	0.982	
GOAJIRO 3	VEN	W ZULIA	128	119	114	3	0	2	0	0	0.013	0.000	0.008	0.979	
GUAHIBO*	VEN	APURE	134	109	107	1	0	1	0	0	0.005	0.000	0.005	0.990	
GUARANI* 2	BRZ	S CATARIN	233	34	34	0	0	0	0	0	0.000	0.000	0.000	1.000	
GUAYMI*	PAN	N W COAST	178	240	240	0	0	0	0	0	0.000	0.000	0.000	1.000	
GUAYO*	SEE	WARAO													
HOPI AND TEWA	USA	NEW MEX	042	123	115	3	5	0	0	0	0.012	0.021	0.000	0.966	
HUASTEC* 1	MEX	VERA CRUZ	024	52	52	0	0	0	0	0	0.000	0.000	0.000	1.000	
ICA	COL	S NEV	131	114	110	3	0	1	0	0	0.013	0.000	0.004	0.982	
IRAPA*	SEE	YUPA													
ISCONAHUA* 2	PRU	R UCAYALI	168	14	14	0	0	0	0	0	0.000	0.000	0.000	1.000	
JIVARO* 2	ECD	ARAPICOS	167	233	233	0	0	0	0	0	0.000	0.000	0.000	1.000	
KARAPALO ANO*2	BRZ	MA GROSSO	201	73	73	0	0	0	0	0	0.000	0.000	0.000	1.000	
KEKCHI 1	GUA	COBAN	173	162	154	4	1	2	1	0	0.016	0.003	0.009	0.972	
KEKCHI* 2	BRH	TOLEDO	176	119	117	2	0	0	0	0	0.008	0.000	0.000	0.992	
LACANDON* 1	MEX	CHIAPAS	170	33	32	1	0	0	0	0	0.015	0.000	0.000	0.985	
LACANDON* 2	MEX	CHIAPAS	171	61	61	0	0	0	0	0	0.000	0.000	0.000	1.000	
MACOITA*	SEE	YUPA													
MACUSHI*	GUY		130	119	119	0	0	0	0	0	0.000	0.000	0.000	1.000	
MAKIRITARE* 1	VEN	S BOLIVAR	130	86	86	0	0	0	0	0	0.000	0.000	0.000	1.000	
MAM 1	GUA	SAN JUAN	171	116	112	3	1	0	0	0	0.013	0.004	0.000	0.982	
MAM 2	GUA	HUEHUETGO	170	24	22	2	0	0	0	0	0.043	0.000	0.000	0.957	
MAPUCHE 2	CHL	LONQUIMAY	163	141	130	9	1	1	0	0	0.032	0.004	0.004	0.959	
MARICOPA*	USA	ARIZ	042	38	38	0	0	0	0	0	0.000	0.000	0.000	1.000	
MOJAVE*	USA	ARIZ	042	117	109	7	0	1	0	0	0.030	0.000	0.004	0.965	

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NASKAPI           CAN   QUEBEC        033       152  101   50    0    1    0    0    0.181   0.000   0.003   0.815
NAVAJO 2          USA   ARIZ          042       106   61   45    0    0    0    0    0.241   0.000   0.000   0.759
NAVAJO 4          USA   ARIZ          063       237  173   61    3    0    0    0    0.138   0.007   0.000   0.854
OTOMI*            MEX   HIDALGO       024        81   76    5    0    0    0    0    0.031   0.000   0.000   0.969
PANARE*           VEN   W BOLIVAR     130        33   33    0    0    0    0    0    0.000   0.000   0.000   1.000
PARIRI*           SEE    YUPA
PEMON*      3P    VEN   LA SABANA     124       287  287    0    0    0    0    0    0.000   0.000   0.000   1.000
  ARECUNA*        VEN   LA SABANA     124        70   70    0    0    0    0    0    0.000   0.000   0.000   1.000
  CAMARACOTO*     VEN   LA SABANA     124       109  109    0    0    0    0    0    0.000   0.000   0.000   1.000
  TAUREPAN*       VEN   LA SABANA     124
PIAROA* 1         VEN   APURE         134       111  111    0    0    0    0    0    0.000   0.000   0.000   1.000
PIMA*             USA   ARIZ          042       489  395   93    0    0    1    0    0.101   0.000   0.001   0.898
PIRO*             PRU   R URUBAMB     168        90   90    0    0    0    0    0    0.000   0.000   0.000   1.000
QUECHUA 1         ECD   NORTHERN      167       232  217    5    0    9    0    1    0.011   0.002   0.022   0.964     C
QUECHUA 2         ECD   CENTRAL       167       372  354   13    1    4    0    0    0.018   0.001   0.005   0.975
QUICHE 1          GUA   S CENTRAL     170       203  188   13    0    2    0    0    0.033   0.000   0.005   0.962
RAMA              NIC   RAMA CAY      175        38   37    1    0    0    0    0    0.013   0.000   0.000   0.987
SANEMA*           SEE    YANOMAMA
SECOYA*           ECD   CUYABENO      167        48   48    0    0    0    0    0    0.000   0.000   0.000   1.000
SHAPARU*          SEE    YUPA
SHIPIBO* 1        PRU   R UCAYALI     168       142  141    0    0    1    0    0    0.000   0.000   0.004   0.996
SIRIONO*          BOL   R MACHUPO     169        27   27    0    0    0    0    0    0.000   0.000   0.000   1.000
SUMO*             NIC   E COAST       175       103  103    0    0    0    0    0    0.000   0.000   0.000   1.000
TAUREPAN          SEE    PEMON
TERRABAS*         COS   TERRABA       177        40   40    0    0    0    0    0    0.000   0.000   0.000   1.000
TICUNA*           PRU   R AMAZON      168       122  122    0    0    0    0    0    0.000   0.000   0.000   1.000
TOTONAC 1         MEX   VERA CRUZ     024       150  142    5    0    3    0    0    0.017   0.000   0.010   0.973
TOTONAC 2         MEX   VERA CRUZ     170        45   41    3    0    1    0    0    0.034   0.000   0.011   0.954
TUNEBO*           COL   COCUY MTS     131       100   98    1    0    1    0    0    0.005   0.000   0.005   0.990
TZELTAL*          MEX   CHIAPAS       170       111  110    1    0    0    0    0    0.005   0.000   0.000   0.995
TZOTZIL* 1        MEX   CHIAPAS       171        80   79    1    0    0    0    0    0.006   0.000   0.000   0.994
TZOTZIL* 2        MEX   CHIAPAS       170        91   89    0    0    2    0    0    0.000   0.000   0.011   0.989
UTE 1             USA   UTAH          163       138  135    3    0    0    0    0    0.011   0.000   0.000   0.989
UTE 2             USA   UTAH          163       104  102    2    0    0    0    0    0.010   0.000   0.000   0.990
WAICA*            SEE    YANOMAMA
WAPISHANA         GUY                 134       119  119    0    0    0    0    0    0.000   0.000   0.000   1.000
WARAO*            VEN   ORINOCO D     134       127  127    0    0    0    0    0    0.000   0.000   0.000   1.000
  GUAYO*          VEN   ORINOCO D     120        81   81    0    0    0    0    0    0.000   0.000   0.000   1.000
  WINIKINA*       VEN   ORINOCO D     120        72   72    0    0    0    0    0    0.000   0.000   0.000   1.000
XAVANTE*    3P    BRZ   MA GROSSO     091       539  539    0    0    0    0    0    0.000   0.000   0.000   1.000
YAGUA*            PRU   R AMAZON      168         9    9    0    0    0    0    0    0.000   0.000   0.000   1.000
YANOMAMA*  10P    VEN   S BOLIVAR     014       572  572    0    0    0    0    0    0.000   0.000   0.000   1.000
  SANEMA*         VEN   S BOLIVAR     134       156  156    0    0    0    0    0    0.000   0.000   0.000   1.000
  WAICA*          VEN   S BOLIVAR     129       141  141    0    0    0    0    0    0.000   0.000   0.000   1.000
YARURO*           VEN   APURE         123       102  102    0    0    0    0    0    0.000   0.000   0.000   1.000
YUMA*             USA   ARIZ          042       182  167   14    0    1    0    0    0.039   0.000   0.003   0.958
YUPA*       3P    VEN   E ZULIA       130       176  176    0    0    0    0    0    0.000   0.000   0.000   1.000
  IRAPA* 2        VEN   E ZULIA       126       138  138    0    0    0    0    0    0.000   0.000   0.000   1.000
  MACOITA*        VEN   E ZULIA       126        78   78    0    0    0    0    0    0.000   0.000   0.000   1.000
  PARIRI*         VEN   E ZULIA       126        74   74    0    0    0    0    0    0.000   0.000   0.000   1.000
  SHAPARU*        VEN   E ZULIA       126        24   24    0    0    0    0    0    0.000   0.000   0.000   1.000
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TABLE 18. THE A. B AND O GENE FREQUENCIES AS DETERMINED BY ANTI-A AND ANTI-B SERA. THE SIGNIFICANT DEPARTURE FROM HARDY-WEINBERG EQUILIBRIUM NOTED IN THE COLUMN HEADED P IS IN EACH CASE OCCASIONED LARGELY BY THE OCCURRENCE OF ONE OR MORE TYPE AB PERSONS IN A SAMPLE WHERE EXPECTATION FOR THIS EVENT IS VERY LOW.

TRIBE OR GROUP	NATION	REGION	REF	SAMPLE SIZE	PHENOTYPES				GENE FREQUENCIES			P
					O	A	B	AB	A	B	O	
ALACALUF 1	CHL	MAGELLAN	074	45	42	3	0	0	0.034	0.000	0.966	
ALACALUF* 2	CHL	MAGELLAN	147	9	9	0	0	0	0.000	0.000	1.000	
ALKUYANA*	SUR		109	70	70	0	0	0	0.000	0.000	1.000	
ANDIDO	ARG	JUJUY	205	209	198	8	0	3	0.027	0.007	0.966	C
ATACAMENOS 2	CHL	CASPANA	074	60	58	0	2	0	0.000	0.017	0.983	
ATACAMENOS 3	CHL	TOCONCE	074	50	47	3	0	0	0.030	0.000	0.970	
AYMARA* 2	BOL	MOCOMOCO	259	100	94	5	1	0	0.025	0.005	0.970	
BAKAIRI*	BRZ	MA GROSSO	146	109	108	1	0	0	0.005	0.000	0.995	
BORORO*	BRZ	MA GROSSO	197	119	119	0	0	0	0.000	0.000	1.000	
BOTOCUDO*	BRZ	MI GERAIS	153	35	35	0	0	0	0.000	0.000	1.000	
CAIUA*	BRZ	MA GROSSO	197	237	237	0	0	0	0.000	0.000	1.000	
CAMAYURA*	BRZ	MA GROSSO	146	60	60	0	0	0	0.000	0.000	1.000	
CANARI	ECD	CANAR	242	1726	1646	53	20	7	0.018	0.008	0.974	C
CARA	ECD	IMBABURA	242	1838	1754	59	16	9	0.019	0.007	0.974	C
CARAJA*	BRZ	ST ISABEL	108	86	86	0	0	0	0.000	0.000	1.000	
CARIB 1	COL	CALDAS	071	540	519	17	4	0	0.016	0.004	0.979	
CARIB 2	SUR		061	177	169	4	4	0	0.011	0.011	0.977	
CARIB* 3	SUR		060	104	104	0	0	0	0.000	0.000	1.000	
CARINA*	VEN	ANZOATEGI	118	170	169	1	0	0	0.003	0.000	0.997	
CAYAPA* 1	ECD	R CAYAPAS	243	62	62	0	0	0	0.000	0.000	1.000	
CHAGUANCO ANO*	ARG	SALTA	181	120	120	0	0	0	0.000	0.000	1.000	
CHINANTEC	MEX	OAXACA	064	21	21	0	0	0	0.000	0.000	1.000	
CHIPAYA 1	BOL	LA PAZ	259	100	94	5	1	0	0.025	0.005	0.970	
CHIPAYA* 2	BOL	SABAYA	258	77	77	0	0	0	0.000	0.000	1.000	
CHIPPEWA	USA	MINN	159	161	141	20	0	0	0.064	0.000	0.936	
CHOL	MEX	CHIAPAS	064	152	135	16	1	0	0.054	0.003	0.943	
CHONTOL	MEX	TARASCO	064	101	88	10	3	0	0.051	0.015	0.933	
CHULUPI* 1	ARG	SALTA	182	55	55	0	0	0	0.000	0.000	1.000	
CHULUPI 2	ARG	SALTA	180	282	269	12	1	0	0.022	0.002	0.976	
COLORADO* 1	ECU	S DOMINGO	243	97	97	0	0	0	0.000	0.000	1.000	
CORA*	MEX	NAYARIT	064	96	93	3	0	0	0.016	0.000	0.984	
CREE*	CAN	ALBERTA	096	33	33	0	0	0	0.000	0.000	1.000	
CUNA* 1	PAN	S BLAS IS	072	89	89	0	0	0	0.000	0.000	1.000	
EMERILLON*	FRG		114	39	39	0	0	0	0.000	0.000	1.000	
GOAJIRO* 1	VEN	W ZULIA	118	152	151	1	0	0	0.003	0.000	0.997	
GOAJIRO* 2	VEN	W ZULIA	193	488	488	0	0	0	0.000	0.000	1.000	
GUARANI* 1	BRZ	R G SUL	216	107	107	0	0	0	0.000	0.000	1.000	
GUATUSO*	COS	ALAJUELA	081	65	65	0	0	0	0.000	0.000	1.000	
HUASTEC 2	MEX	VERA CRUZ	064	76	72	0	4	0	0.000	0.027	0.973	
HUICHOL	MEX	JALISCO	064	71	71	0	0	0	0.000	0.000	1.000	
IRAPA* 1	SEE	YUPA										
ISCONAHUA* 1	PRU	R UCAYALI	043	16	16	0	0	0	0.000	0.000	1.000	
JIVARO* 1	ECD	ARAPICO	243	111	111	0	0	0	0.000	0.000	1.000	
KARAPALO* 1	BRZ	MA GROSSO	146	81	81	0	0	0	0.000	0.000	1.000	
KUAIKER	COL	PUTUMAYO	136	172	165	4	3	0	0.012	0.009	0.979	
KUTCHIN	USA	ALASKA	145	114	113	0	1	0	0.000	0.004	0.996	
LLOREOS*	COL	R ATRATO	253	81	81	0	0	0	0.000	0.000	1.000	
MACA	PAR	CHACO	265	111	108	1	2	0	0.005	0.009	0.986	
MACOITA* 1	SEE	YUPA										
MACU*	BRZ	AMAZONAS	031	9	9	0	0	0	0.000	0.000	1.000	
MAM POOL 3	GUA	W AND SW	262	70	65	3	2	0	0.022	0.014	0.963	
MAPUCHE 1	CHL	LONQUIMAY	074	148	143	5	0	0	0.017	0.000	0.983	
MATACO*	ARG	SALTA	181	135	130	5	0	0	0.019	0.000	0.981	
MAYA 1	MEX	YUCATAN	095	223	218	3	1	1	0.009	0.004	0.986	C
MAYA 2	MEX	YUCATAN	218	126	124	2	0	0	0.008	0.000	0.992	
MAYA* 3	GUA	F PETEN	263	40	40	0	0	0	0.000	0.000	1.000	

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MAZATEC          MEX  OAXACA        064   136  127   8   1   0   0.030  0.004  0.965
MEHINAKO*        BRZ  MA GROSSO     146    47   47   0   0   0   0.000  0.000  1.000
MOCETINES*       BOL  AMAZONIA      223    76   75   1   0   0   0.007  0.000  0.993
NAHUA 1          MEX  PUEBLA        229    50   49   1   0   0   0.010  0.000  0.990
NAHUA 2          MEX  PUEBLA        064    42   38   4   0   0   0.049  0.000  0.951
NAHUA 3          MEX  VERA CRUZ     064   141  127  13   0   1   0.051  0.004  0.945   C
NAVAJO* 1        USA  NEW MEX       218   112  111   1   0   0   0.004  0.000  0.996
NOOTKA* 1        CAN  N W COAST     089    61   60   1   0   0   0.008  0.000  0.992
NOOTKA 2         CAN  N W COAST     102   276  267   9   0   0   0.016  0.000  0.984
OAYANA*          FRG                114   100  100   0   0   0   0.000  0.000  1.000
OMAHA            USA  S DAK         157   168  148  18   1   1   0.058  0.006  0.935   B
ONA*             CHL  MAGELLAN      147     5    5   0   0   0   0.000  0.000  1.000
OYAMPI*          FRG                114    99   98   1   0   0   0.005  0.000  0.995
PAEZ*            COL  CALDAS        071    50   49   1   0   0   0.010  0.000  0.990
PALIKOUR*        FRG                114    75   74   0   1   0   0.000  0.007  0.993
PALTA            ECD  LOJA          242   568  546  13   8   1   0.012  0.008  0.979   B
PANZALEO         ECD  COTOPAXI      242  1446 1376  49  20   1   0.017  0.007  0.975
PAPAGO           USA  ARIZ          041   600  563  37   0   0   0.031  0.000  0.969
PEHU[illegible]CHE        CHL  LONQUIMAY     074   182  174   6   2   0   0.017  0.006  0.977
PIAROA* 2        VEN  AMAZONAS      134    24   24   0   0   0   0.000  0.000  1.000
PIJAO* 1         COL  COYAIMA       213   439  423  15   0   1   0.018  0.001  0.980   C
PIJAO* 2         COL  NATAGAIMA     213   281  281   0   0   0   0.000  0.000  1.000
POCOMAM ANO      GUA  CENTRAL       262   132  121   7   4   0   0.027  0.015  0.957
PURUHA           ECD  CHIMBRAZO     242  1410 1334  63  11   2   0.023  0.005  0.972   B
QUICHE 2         GUA  S CENTRAL     262   129  127   1   1   0   0.004  0.004  0.991
RIONEGRINO*      SEE   YUPA
SERI*            MEX  BAJA CAL      029   128  128   0   0   0   0.000  0.000  1.000
SHIPIBO* 2       PRU  R UCAYALI     043    70   70   0   0   0   0.000  0.000  1.000
TARIANA*         BRZ  R NEGRO       031    48   48   0   0   0   0.000  0.000  1.000
TOBA* 1          ARG  SALTA         182   194  191   3   0   0   0.008  0.000  0.992
TOBA* 2          ARG  CHACO         204   402  396   5   0   1   0.007  0.001  0.991   C
TOTONAC 3        MEX  PUEBLA        064    79   70   9   0   0   0.059  0.000  0.941
TUCANO* 1        BRZ  AMAZONAS      031   180  180   0   0   0   0.000  0.000  1.000
TUCANO 2         BRZ  AMAZONAS      186   131  127   3   1   0   0.011  0.004  0.984
URU*             PRU  TITICACA      267    27   27   0   0   0   0.000  0.000  1.000
WAURA*           BRZ  MA GROSSO     146    80   80   0   0   0   0.000  0.000  1.000
YARAPANA*        VEN  MANAPIARE     134    14   14   0   0   0   0.000  0.000  1.000
YAMANA*          CHL  NAVARINO      147    20   20   0   0   0   0.000  0.000  1.000
YUMBO*           ECD  AMAZONAS      243   944  925  17   2   0   0.009  0.001  0.990
YUPA
  IRAPA* 1       VEN  E ZULIA       193   161  161   0   0   0   0.000  0.000  1.000
  MACOITA* 1     VEN  E ZULIA       195   117  117   0   0   0   0.000  0.000  1.000
  RIONEGRINO*    VEN  E ZULIA       194   125  125   0   0   0   0.000  0.000  1.000
ZAPOTEC          MEX  OAXACA        064   106   96   7   3   0   0.034  0.014  0.952
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TABLE 2A. THE MS, MZ, NS, AND NZ GENE FREQUENCIES, AS DETERMINED BY ANTI-M, ANTI-N, ANTI-S, AND ANTI-Z SERA. IN THE TABLE, READ 'SMALL S' FOR 'Z'. FOR CLARITY IN THE COMPUTER PRINT-OUT, WHERE ALTERNATIVE PAIRED REACTIONS EXIST, OR WHERE A PHENOTYPE IS DUE TO HOMOZYGOSITY, THE PHENOTYPE IS WRITTEN BY A DOUBLE LETTER DESIGNATION, THUS MMZZ FOR MZ.

TRIBE OR GROUP	NATION	REGION	REF	SAMPLE SIZE	PHENOTYPES MMSS	MMZZ	MMSZ	MNSS	MNZZ	MNSZ	NNSS	NNZZ	NNSZ	GENE FREQUENCIES MS	MZ	NS	NZ	P
ACAWAIO*	GUY		130	90	1	27	12	1	26	13	0	9	1	0.133	0.533	0.033	0.300	
AGUARUNA*	PRU	R MARANON	168	151	8	17	36	10	31	28	5	7	9	0.253	0.378	0.141	0.227	
ALACALUF 3	CHL	MAGELLAN	164	44	0	14	6	0	16	3	0	5	0	0.102	0.568	0.000	0.329	
ARECUNA*	SEE	PEMON																
ATACAMENOS 1	CHL	ANTOFAGST	164	80	4	34	15	0	12	9	0	6	0	0.200	0.593	0.000	0.206	
AYMARA* 1	PRU	PUNO	028	58	7	11	7	4	13	6	1	7	2	0.251	0.377	0.085	0.286	
AYMARA* 3	PRU	PUNO	168	93	3	14	14	1	33	15	0	13	0	0.190	0.406	0.009	0.394	
AYMARA* 4	BOL	3 AREAS P	169	503	13	126	113	7	128	61	0	46	9	0.193	0.502	0.029	0.275	
BARI*	VEN	W ZULIA	132	119	5	36	18	6	36	11	1	3	3	0.165	0.553	0.071	0.211	
BRIBRI*	COS	SALITRE	177	50	2	8	11	6	12	7	0	2	2	0.247	0.423	0.113	0.217	
CABECAR*	COS	UJARRAS	177	62	10	15	32	0	0	5	0	0	0	0.419	0.540	0.040	0.000	
CAKCHIQUEL 1	GUA	L ALTITLN	171	150	9	33	20	14	37	26	4	1	6	0.203	0.467	0.150	0.180	
CAKCHIQUEL 3	GUA	SUMPANGO	268	137	3	26	29	9	23	38	2	2	5	0.200	0.478	0.165	0.156	
CAMARACOTO*	SEE	PEMON																
CAMPA*	PRU	URUBAMBA	168	89	20	9	23	14	2	18	2	0	1	0.457	0.317	0.183	0.042	
CAYAPA* 2	ECD	R CAYAPAS	167	244	32	67	67	2	44	27	0	5	0	0.324	0.505	0.008	0.162	
CHACOBO*	BOL	R YATA	169	14	6	1	0	3	0	2	2	0	0	0.535	0.143	0.321	0.000	
CHAMA*	BOL	R MADIDI	169	30	5	13	12	0	0	0	0	0	0	0.367	0.633	0.000	0.000	
CHEROKEE*	USA	N CARLINA	209	78	14	16	20	4	11	10	2	1	0	0.369	0.432	0.079	0.119	
CHIPPEWA	USA	MINN	159	161	12	23	51	1	33	26	1	9	5	0.302	0.419	0.040	0.239	
CHOCO*	PAN	DARIEN	178	80	10	12	11	0	22	13	1	6	5	0.256	0.375	0.062	0.306	
COLORADO* 2	ECD	S DOMINGO	167	36	1	19	4	0	12	0	0	0	0	0.083	0.750	0.000	0.167	
CUIVA*	VEN	APURE	134	82	0	48	0	0	31	0	0	3	0	0.000	0.774	0.000	0.226	
CUNA* 2	PAN	S BLAS IS	178	388	34	83	80	15	82	66	5	21	2	0.268	0.449	0.062	0.220	A
GOAJIRO 3	VEN	W ZULIA	128	119	18	17	27	10	18	18	0	8	3	0.362	0.351	0.074	0.212	
GUAHIBO*	VEN	APURE	134	109	0	76	10	0	21	0	0	2	0	0.046	0.838	0.000	0.115	
GUAYMI*	PAN	N W COAST	178	240	19	43	54	6	53	44	0	17	4	0.280	0.417	0.036	0.266	
ICA	COL	S NEV	131	113	11	39	36	6	5	14	1	0	1	0.294	0.577	0.091	0.037	
IRAPA* 2	SEE	YUPA																
ISCONAHUA* 1	PRU	R UCAYALI	043	16	12	1	3	0	0	0	0	0	0	0.844	0.156	0.000	0.000	
ISCONAHUA* 2	PRU	R UCAYALI	168	14	10	0	4	0	0	0	0	0	0	0.857	0.143	0.000	0.000	
JIVARO* 2	ECD	ARAPICOS	167	233	33	54	81	2	34	26	0	3	0	0.371	0.482	0.008	0.138	
KEKCHI 1	GUA	COBAN	173	162	8	31	33	7	40	28	0	12	3	0.236	0.439	0.055	0.269	
KEKCHI* 2	BRH	TOLEDO	176	119	1	29	33	6	21	18	0	4	7	0.205	0.512	0.098	0.184	
KUTCHIN	USA	ALASKA	145	92	3	36	16	7	14	13	0	1	2	0.175	0.607	0.102	0.115	
LACANDON* 2	MEX	CHIAPAS	171	61	0	16	3	0	18	7	0	17	0	0.082	0.434	0.000	0.483	
MACOITA*	SEE	YUPA																
MACUSHI*	GUY		130	119	10	24	14	2	35	20	0	9	5	0.215	0.428	0.050	0.307	
MAKIRITARE* 1	VEN	S BOLIVAR	130	86	9	9	24	11	7	18	2	1	5	0.351	0.346	0.177	0.125	
MAM 1	GUA	SAN JUAN	171	116	3	27	20	7	28	16	1	6	8	0.172	0.478	0.113	0.237	
MAPUCHE 2	CHL	LONQUIMAY	164	141	3	44	16	4	43	20	0	11	0	0.137	0.547	0.041	0.275	
NASKAPI	CAN	QUEBEC	033	152	27	40	65	0	8	10	0	2	0	0.424	0.503	0.000	0.072	
NAVAJO 4	USA	ARIZ	063	237	20	63	81	6	31	24	0	9	3	0.305	0.516	0.033	0.146	
PANARE*	VEN	W BOLIVAR	130	33	1	7	3	0	12	4	0	5	1	0.124	0.451	0.027	0.397	
PARIRI*	SEE	YUPA																
PEMON* 3P	VEN	LA SABANA	124	287	1	75	24	5	97	38	3	23	21	0.074	0.518	0.102	0.305	
ARECUNA*	VEN	LA SABANA	124	70	1	23	7	0	21	9	0	4	5	0.086	0.571	0.078	0.264	
CAMARACOTO*	VEN	LA SABANA	124	109	0	24	10	2	33	17	2	13	8	0.081	0.468	0.117	0.333	
TAUREPAN*	VEN	LA SABANA	124	108	0	28	7	3	43	12	1	6	8	0.060	0.531	0.102	0.306	
PIAROA* 1	VEN	APURE	134	109	14	31	40	3	13	7	0	1	0	0.349	0.536	0.023	0.092	
PIRO*	PRU	R URUBAMB	168	90	9	18	18	3	16	16	2	1	7	0.257	0.437	0.126	0.179	
QUECHUA 1	ECD	NORTHERN	167	232	16	67	70	0	44	23	0	8	4	0.261	0.541	0.017	0.180	
QUECHUA 2	ECD	CENTRAL	167	372	27	113	94	2	84	35	0	15	2	0.244	0.547	0.010	0.198	
RAMA	NIC	RAMA CAY	175	37	2	3	6	3	7	5	1	6	4	0.220	0.280	0.145	0.354	
SANEMA*	SEE	YANOMAMA																

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SECOYA*            ECD   CUYABENO      167      48    4   24   12    0    5    3    0    0    0  0.235 0.681 0.004 0.079
SHAPARU*           SEE    YUPA
SHIPIBO* 1         PRU   R UCAYALI     168     142   12   26   30   16   22   25    4    1    6  0.278 0.422 0.162 0.137
SHIPIBO* 2         PRU   R UCAYALI     043      70    6    8    9    9    6   15    7    2    8  0.248 0.295 0.294 0.163
SIRIONO*           BOL   R MACHUPO     169      27    0    0    1    0    4   11    0    0   11  0.038 0.277 0.387 0.297
SUMO*              NIC   E COAST       175     103   66    0   17    4    2   13    1    0    0  0.802 0.096 0.032 0.069
TAUREPAN*          SEE    PEMON
TERRABAS*          COS   TERRABA       177      40    1   13    7    1   10    4    1    1    2  0.142 0.569 0.096 0.192
TICUNA*            PRU   R AMAZON      168     122    2   68   12    0   27    6    1    6    0  0.077 0.729 0.022 0.171
TUNEBO*            COL   COCUY MTS     131     100   14   13   12   12   15   21    0   11    2  0.341 0.288 0.093 0.277
TZOTZIL* 1         MEX   CHIAPAS       171      80    7   11   10    2   19   14    2    9    6  0.221 0.347 0.104 0.327
WAICA*             SEE    YANOMAMA
WAPISHANA          GUY                 134     119   26   21   35    9    6   21    0    0    1  0.444 0.395 0.088 0.072
WARAO*             VEN   ORINOCO D     134     127    3   15   10    6   11   29   12   14   27  0.120 0.282 0.304 0.293
XAVANTE*     3P    BRZ   MA GROSSO     091     537   91   85  147   32   75   78    5    7   17  0.382 0.391 0.081 0.145
YAGUA*             PRU   R AMAZON      168       9    0    8    1    0    0    0    0    0    0  0.056 0.944 0.000 0.000
YANOMAMA*   10P    VEN   S BOLIVAR     014     572    8  215   57    3  175   51    3   40   20  0.085 0.603 0.052 0.259
  SANEMA*          VEN   S BOLIVAR     134     156    2   49   15    0   57    5    1   25    2  0.073 0.548 0.017 0.361
  WAICA*           VEN   S BOLIVAR     129     141    1   62   27    1   24   17    1    5    3  0.125 0.661 0.063 0.150
YARURO*            VEN   APURE         123     102    3   42   21    1   22    5    0    5    3  0.152 0.632 0.029 0.186
YUPA*        3P    VEN   E ZULIA       130     176   32   42   36   14   20   23    0    6    3  0.365 0.421 0.073 0.140
  IRAPA* 2         VEN   E ZULIA -     126     138    3   45   10    1   40    8    0   27    4  0.081 0.516 0.027 0.375
  MACOITA* 2       VEN   E ZULIA       126      78    7   14   19    4   13   13    0    6    2  0.299 0.405 0.060 0.235
  PARIRI*          VEN   E ZULIA       126      74   16   18   18    8    3   10    0    0    1  0.411 0.432 0.109 0.047
  SHAPARU*         VEN   E ZULIA       126      24    5    7    6    0    4    2    0    0    0  0.375 0.500 0.000 0.125
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TABLE 28. THE MS, MZ, NS, AND NZ GENE FREQUENCIES, AS DETERMINED BY ANTI-M, ANTI-N, AND ANTI-S SERA. IN THE TABLE, READ 'SMALL S' FOR 'Z'. FOR CLARITY IN THE COMPUTER PRINT-OUT, WHERE ALTERNATIVE PAIRED REACTIONS EXIST, OR WHERE A PHENOTYPE IS DUE TO HOMOZYGOSITY, THE PHENOTYPE IS WRITTEN WITH A DOUBLE LETTER DESIGNATION, THUS MMZZ FOR MZ.

TRIBE OR GROUP	NATION	REGION	REF	SAMPLE SIZE	PHENOTYPES						GENE FREQUENCIES				P
					MMS	MMZZ	MNS	MNZZ	NNS	NNZZ	MS	MZ	NS	NZ	
ALACALUF 1	CHL	MAGELLAN	074	45	7	14	2	17	0	5	0.107	0.571	0.000	0.322	
ATHABASCAN 1	USA	ALASKA	062	78	16	56	1	5	0	0	0.114	0.848	0.002	0.036	
CARAJA*	BRZ	ST ISABEL	108	51	14	15	3	2	10	7	0.175	0.441	0.140	0.243	
DIEGUENO	USA	CAL	202	39	17	5	8	7	0	2	0.398	0.358	0.000	0.243	
HOPI AND TEWA	USA	NEW MEX	042	52	1	9	5	25	2	10	0.029	0.452	0.051	0.467	
KALAPALO AND*2	BRZ	MA GROSSO	201	74	8	6	35	24	0	1	0.223	0.364	0.129	0.283	
MAM POOL 3	GUA	W AND SW	262	70	6	26	9	22	4	3	0.063	0.615	0.084	0.238	
MARICOPA*	USA	ARIZ	042	19	0	4	4	9	0	2	0.000	0.553	0.105	0.342	
MAYA* 3	GUA	E PETEN	263	120	40	20	27	24	5	4	0.290	0.421	0.078	0.210	
NAVAJO* 2	USA	ARIZ	042	25	0	4	4	16	0	1	0.000	0.560	0.080	0.360	
PIMA*	USA	ARIZ	042	172	22	56	27	59	2	6	0.110	0.593	0.051	0.245	
POCOMAM AND	GUA	CENTRAL	262	80	24	19	11	19	3	4	0.228	0.496	0.048	0.227	
QUICHE 2	GUA	S CENTRAL	262	80	22	21	18	14	2	3	0.230	0.507	0.081	0.181	
TOTONACA 2	MEX	VERA CRUZ	170	45	18	17	3	5	1	1	0.257	0.610	0.028	0.105	
TZELTAL*	MEX	CHIAPAS	170	22	4	5	11	1	1	0	0.173	0.508	0.283	0.035	
YABARANA*	VEN	MANAPIARE	134	14	0	11	0	3	0	0	0.000	0.893	0.000	0.107	

TABLE 2C. THE M AND N GENE FREQUENCIES, AS DETERMINED BY ANTI-M AND ANTI-N SERA.

TRIBE OR GROUP	NATION	REGION	REF	SAMPLE SIZE	PHENOTYPES			GENE FREQUENCIES		P
					MM	MN	NN	M	N	
APACHE* 1	USA	ARIZ	042	176	97	59	20	0.719	0.281	
APACHE 2	USA	NEW MEX	090	108	76	30	2	0.843	0.157	
AWEIKOMA* ANO	BRZ	S CATARIN	233	102	29	55	18	0.554	0.446	
CAINGANG	BRZ	R G SUL	231	205	103	86	16	0.712	0.288	
CAIUA*	BRZ	MA GROSSO	197	134	107	26	1	0.896	0.104	
CAKCHIQUEL* 2	GUA	SOLALA	170	9	3	5	1	0.611	0.389	
CHAMULA*	MEX	CHIAPAS	024	63	40	21	2	0.802	0.198	
CHINANTEC	MEX	OAXACA	064	20	11	6	3	0.700	0.300	
CHOL	MEX	CHIAPAS	064	152	71	71	10	0.701	0.299	
CHONTOL	MEX	TARASCO	064	101	53	34	14	0.693	0.307	
CHULUPI2	ARG	SALTA	180	282	231	32	19	0.876	0.124	C
CORA	MEX	NAYARIT	064	96	55	36	5	0.760	0.240	
EMERILLON*	FRG		114	26	11	13	2	0.673	0.327	
GUARANI* 2	BRZ	S CATARNA	233	34	9	19	6	0.544	0.456	
GUATUSO*	COS	ALAJUELA	081	65	20	29	16	0.531	0.469	
GUAYO*	SEE	WARAO								
HUASTEC* 1	MEX	VERA CRUZ	024	49	43	4	2	0.918	0.082	B
HUASTEC 2	MEX	VERA CRUZ	064	75	42	29	4	0.753	0.247	
HUICHOL	MEX	JALISCO	064	71	38	30	3	0.746	0.254	
IRAPA* 1	SEE	YUPA								
LACANDON* 1	MEX	CHIAPAS	170	33	23	8	2	0.818	0.182	
MACOITA* 1	SEE	YUPA								
MAM 2	GUA	HUEHUETNG	170	24	13	11	0	0.771	0.229	
MAZATEC	MEX	OAXACA	064	136	64	62	10	0.699	0.301	
MOCETENES*	ROL	AMAZONIA	223	76	44	26	6	0.750	0.250	
MOJAVE ANO*	USA	ARIZ	042	116	62	44	10	0.724	0.276	
NAHUA 2	MEX	PUERLA	064	41	25	12	4	0.756	0.244	
NAHUA 3	MEX	VERA CRUZ	064	141	83	51	7	0.770	0.230	
NAVAJO 2	USA	ARIZ	042	104	30	67	7	0.611	0.389	A
NOOTKA 2	CAN	N W COAST	102	276	213	33	30	0.832	0.168	C
OAYANA*	FRG		114	100	59	33	8	0.755	0.245	
OTOMI*	MEX	HIDALGO	024	81	60	20	1	0.864	0.136	
OYAMPI*	FRG		114	81	22	49	10	0.574	0.426	
PALIKOUR*	FRG		114	75	23	42	10	0.587	0.413	
QUICHE 1	GUA	S CENTRAL	170	203	112	74	17	0.734	0.266	
RIONEGRINO*	SEE	YUPA								
TOTONAC 1	MEX	VERA CRUZ	024	71	45	21	5	0.782	0.218	
TOTONAC 3	MEX	PUERLA	064	79	50	25	4	0.791	0.209	
TUCANO* 1	BRZ	AMAZONAS	031	68	33	29	6	0.699	0.301	
TUCANO 2	BRZ	AMAZONAS	186	75	56	2	17	0.760	0.240	C
TZOTZIL* 2	MEX	CHIAPAS	170	91	53	30	8	0.747	0.253	
UTE 2	USA	UTAH	161	104	61	36	7	0.760	0.240	
WARAO										
GUAYO*	VEN	ORINOCO	120	81	23	36	22	0.506	0.494	
WINIKINA*	VEN	ORINOCO	120	72	11	31	30	0.368	0.632	
WINIKINA*	SEE	WARAO								
YUMA*	USA	ARIZ	042	180	97	64	19	0.717	0.283	
YUPA										
IRAPA* 1	VEN	E ZULIA	193	161	74	54	33	0.627	0.373	A
MACOITA* 1	VEN	E ZULIA	193	117	52	49	16	0.654	0.346	
RIONEGRINO*	VEN	E ZULIA	194	125	40	66	19	0.584	0.416	
ZAPOTEC	MEX	OAXACA	064	105	51	41	13	0.681	0.319	

TABLE 3A. THE RH GENE FREQUENCIES AS DETERMINED WITH FIVE ANTISERA (ANTI-C, ANTI-'LITTLE C', ANTI-D, ANTI-E, AND ANTI-'LITTLE E'). BECAUSE THE EVIDENCE FOR THE ABSENCE OF A PARTICULAR CHROMOSOME IS IN SOME SERIES VERY WEAK, THE FORTRAN PROGRAM HAS BEEN WRITTEN ON THE ASSUMPTION THAT ALL THE RH CHROMOSOMES MAY BE PRESENT IN ANY GIVEN POPULATION. THIS RESULTS IN SOME POPULATIONS IN THE ASSIGNMENT OF SMALL POSITIVE FREQUENCIES TO CHROMOSOMES WHICH ARE PROBABLY ABSENT. NO TESTS FOR DEPARTURE FROM HARDY-WEINBERG PROPORTIONS HAVE BEEN PERFORMED.

TRIBE OR GROUP	NATION	REGION	REF	SAMPLE SIZE		PHENOTYPES												GENE FREQUENCIES							
					ANTI-C ANTI-c ANTI-D ANTI-E ANTI-e	+ - - - +	+ + - - +	- + - - +	+ - + - +	+ + + - +	- + + - +	+ - + + +	+ - + + -	+ + + + +	+ + + + -	- + + + -	- + + + +	C D E	C D e	c D E	c D e	C d E	C d e	c d E	c d e
ACAWAIO.	GUY		130	90		0	0	0	29	0	0	7	1	30	6	16	1	0.084	0.525	0.383	0.000	0.000	0.001	0.000	0.006
AGUARUNA.	PRU	R MARANON	168	151		0	0	0	35	18	0	0	0	67	0	26	5	0.000	0.513	0.410	0.076	0.000	0.000	0.000	0.000
ALACALUF	CHL	MAGELLAN	074	45		0	0	0	8	4	0	0	0	14	1	18	0	0.011	0.377	0.563	0.000	0.000	0.000	0.002	0.045
ALACALUF 3	CHL	MAGELLAN	163	44		0	0	0	7	0	0	0	0	18	0	18	1	0.000	0.364	0.625	0.000	0.000	0.000	0.000	0.011
APACHE. 2	USA	NEW MEX	090	105		0	0	0	24	5	1	3	0	45	2	17	8	0.000	0.478	0.422	0.073	0.026	0.000	0.000	0.000
ARECUNA.	SEE	PEMON																							
ATACAMENOS 1	CHL	ANTOFAGST	164	80		0	0	0	4	0	0	8	4	40	5	16	3	0.136	0.341	0.494	0.006	0.000	0.004	0.000	0.018
ATHABASCAN 1	USA	ALASKA	062	78		0	1	0	7	1	0	2	0	34	3	23	7	0.034	0.258	0.574	0.000	0.000	0.073	0.000	0.060
AWEIKOMA ANO.	BRZ	S CATARNA	233	83		0	0	0	25	9	1	2	1	29	5	10	1	0.059	0.537	0.327	0.077	0.000	0.000	0.000	0.000
AYMARA. 1	PRU	PUNO	028	58		0	0	0	11	0	0	3	0	32	1	10	1	0.035	0.490	0.465	0.005	0.000	0.000	0.000	0.004
AYMARA. 2	BOL	MOCOMOCO	259	100		0	0	0	8	3	1	5	1	45	15	13	9	0.124	0.330	0.460	0.085	0.000	0.000	0.000	0.000
AYMARA. 3	PRU	PUNO	168	93		0	0	0	19	0	0	2	0	45	2	25	0	0.000	0.457	0.521	0.000	0.021	0.000	0.000	0.000
AYMARA. 4	BOL	3 AREAS P	169	502		0	0	0	65	5	0	20	3	204	13	189	3	0.039	0.357	0.595	0.000	0.000	0.000	0.000	0.008
BARI.	VEN	W ZULIA	132	126		0	0	0	73	0	1	2	0	39	3	8	0	0.013	0.370	0.185	0.254	0.005	0.125	0.047	0.000
BRIBRI.	COS	SALITRE	177	50		0	0	0	12	0	0	0	0	20	0	11	7	0.000	0.437	0.489	0.000	0.000	0.003	0.000	0.069
CABECAR.	COS	UJARRAS	177	62		0	0	0	17	9	0	0	0	25	0	6	5	0.000	0.547	0.339	0.113	0.000	0.000	0.000	0.000
CAKCHIQUEL 1	GUA	L ALTITLN	171	150		0	0	0	40	5	0	8	0	65	4	24	4	0.001	0.524	0.402	0.031	0.041	0.000	0.000	0.000
CAKCHIQUEL 2	GUA	SOLOLA	170	9		0	0	0	4	0	0	2	0	3	0	0	0	0.036	0.721	0.109	0.000	0.075	0.000	0.058	0.000
CAKCHIQUEL 3	GUA	SUMPANGO	268	137		0	0	0	57	12	0	1	0	47	0	19	1	0.004	0.634	0.314	0.000	0.000	0.000	0.000	0.047
CAMARACOTO.	SEE	PEMON																							
CAMPA.	PRU	R URUBMBA	168	89		0	0	0	8	0	0	9	4	42	9	17	0	0.146	0.376	0.477	0.000	0.000	0.000	0.000	0.000
CARAJA.	BRZ	S ISABEL	108	52		0	0	0	18	9	1	0	0	21	1	2	0	0.000	0.631	0.249	0.107	0.011	0.001	0.000	0.000
CAYAPA.	ECD	R CAYAPAS	167	244		0	0	0	110	0	0	0	0	101	0	32	1	0.000	0.657	0.340	0.000	0.000	0.000	0.000	0.002
CHACOBO.	BOL	R YATA	169	14		0	0	0	1	0	0	2	1	4	3	3	0	0.250	0.286	0.463	0.000	0.000	0.000	0.000	0.000
CHAMA.	BOL	R MADIDI	169	30		0	0	0	8	0	0	1	0	17	0	3	1	0.001	0.565	0.398	0.017	0.016	0.000	0.002	0.000
CHEROKEE.	USA	N CARLINA	209	78		0	0	0	29	4	0	7	0	27	2	7	2	0.001	0.611	0.286	0.041	0.060	0.000	0.000	0.000
CHINANTEC	MEX	OAXACA	064	18		0	0	0	4	1	0	3	1	4	0	3	2	0.149	0.434	0.323	0.000	0.000	0.000	0.000	0.093
CHIPAYA 1	BOL	LA PAZ	259	100		0	0	0	8	3	1	5	1	45	15	13	9	0.124	0.330	0.460	0.085	0.000	0.000	0.000	0.000
CHIPAYA. 2	BOL	SABAYA	258	66		0	0	0	4	0	0	4	1	10	9	34	4	0.116	0.164	0.686	0.000	0.000	0.000	0.000	0.033
CHOCO.	PAN	DARIEN	178	80		0	0	0	33	2	1	1	0	32	1	2	8	0.000	0.630	0.280	0.076	0.014	0.000	0.000	0.000
CHOL	MEX	CHIAPAS	064	152		0	0	0	31	14	3	3	3	59	3	25	11	0.044	0.448	0.400	0.107	0.000	0.000	0.000	0.000
CHONTOL	MEX	TABASCO	066	100		0	0	0	26	12	3	5	2	25	2	20	5	0.060	0.464	0.355	0.120	0.000	0.000	0.000	0.000
COLORADO. 2	ECD	S DOMINGO	167	36		0	0	0	18	1	0	0	0	14	0	3	0	0.000	0.707	0.278	0.000	0.000	0.000	0.000	0.014
CORA.	MEX	NAYARIT	064	96		0	0	0	33	8	1	1	1	24	2	21	5	0.027	0.514	0.379	0.079	0.000	0.000	0.000	0.000
CUIVA.	VEN	APURE	134	82		0	0	0	2	0	0	0	0	37	3	40	0	0.000	0.246	0.731	0.000	0.018	0.004	0.000	0.000
CUNA. 2	PAN	SAN BLAS	178	388		0	0	0	203	8	0	10	0	128	1	34	4	0.014	0.710	0.259	0.001	0.000	0.000	0.000	0.015
GOAJIRO 3	VEN	W ZULIA	128	119		0	0	0	58	11	1	2	0	41	1	4	1	0.000	0.713	0.213	0.060	0.013	0.000	0.000	0.000
GUAHIBO.	VEN	APURE	134	109		0	0	0	19	1	0	14	0	48	3	13	11	0.084	0.456	0.398	0.048	0.000	0.000	0.000	0.013
GUARANI. 2	BRZ	S CATARNA	233	31		0	0	0	17	5	0	0	2	6	0	1	0	0.071	0.718	0.120	0.000	0.000	0.000	0.003	0.087
GUATUSO.	COS	ALAJUELA	081	65		0	0	0	10	20	1	2	1	0	5	19	7	0.069	0.323	0.380	0.205	0.000	0.000	0.000	0.022
GUAYMI.	PAN	N W COAST	178	240		0	0	0	179	9	0	4	0	45	0	3	0	0.009	0.865	0.105	0.000	0.000	0.000	0.001	0.019
HUASTEC 2	MEX	VERA CRUZ	064	52		0	0	1	13	1	1	2	0	24	2	7	1	0.041	0.344	0.389	0.159	0.000	0.002	0.002	0.061
HUICHOL	MEX	JALISCO	064	72		0	0	0	17	8	1	2	0	25	3	10	6	0.000	0.474	0.371	0.115	0.039	0.000	0.000	0.000
ICA	COL	S NEV	131	113		0	0	1	46	35	11	0	0	10	0	5	5	0.000	0.605	0.111	0.201	0.000	0.000	0.000	0.082
IRAPA.	SEE	YUPA																							
ISCONAHUA. 1	PRU	R UCAYALI	043	16		0	0	0	2	2	0	1	0	2	0	6	3	0.033	0.279	0.529	0.002	0.000	0.000	0.000	0.156
ISCONAHUA. 2	PRU	R UCAYALI	168	14		0	0	0	0	1	0	1	0	3	1	5	3	0.000	0.166	0.594	0.155	0.084	0.000	0.000	0.000
JIVARO. 2	ECD	ARAPICOS	167	233		0	0	0	73	22	2	0	0	83	2	34	17	0.000	0.537	0.364	0.093	0.005	0.000	0.000	0.000
KEKCHI 1	GUA	COBAN	173	162		0	0	0	46	4	1	4	0	67	5	28	7	0.000	0.453	0.415	0.068	0.030	0.000	0.000	0.033
KEKCHI. 2	BRH	TOLEDO	176	119		0	0	0	37	2	0	1	0	62	1	14	2	0.009	0.582	0.391	0.003	0.000	0.000	0.000	0.014
KUTCHIN	USA	ALASKA	145	92		0	0	0	4	3	0	0	0	27	1	54	3	0.006	0.206	0.754	0.001	0.000	0.000	0.000	0.032

LACANDON* 1		MEX	CHIAPAS	170	33	0	0	0	4	0	0	0	0	14	1	13	1	0.000	0.332	0.636	0.015	0.015	0.001	0.000	0.000
LACANDON* 2		MEX	CHIAPAS	171	60	0	0	0	17	0	0	2	0	25	1	12	3	0.007	0.507	0.441	0.026	0.018	0.000	0.000	0.000
MACOITA*		SEE	YUPA																						
MACUSHI*		GUY		130	119	0	0	0	34	1	0	15	3	40	3	23	0	0.101	0.520	0.373	0.000	0.000	0.000	0.001	0.004
MAKIRITARE* 1		VEN	S BOLIVAR	130	86	0	0	0	10	6	1	1	0	39	2	22	5	0.000	0.382	0.521	0.077	0.019	0.000	0.000	0.000
MAM 1		GUA	SAN JUAN	171	116	0	0	1	43	2	0	6	0	48	2	14	0	0.022	0.611	0.336	0.000	0.013	0.000	0.000	0.017
MAM 2		GUA	HUEHUETGO	170	24	0	0	0	7	1	0	0	0	10	0	3	3	0.000	0.520	0.396	0.083	0.000	0.000	0.000	0.000
MAPUCHE 2		CHL	LONQUIMAY	163	140	0	0	0	40	16	0	0	0	56	0	22	6	0.000	0.541	0.379	0.069	0.000	0.000	0.000	0.010
MAYA* 3		GUA	E PETEN	263	40	0	0	0	17	2	0	1	4	8	0	7	1	0.115	0.559	0.285	0.000	0.000	0.000	0.000	0.040
MAZATEC		MEX	OAXACA	064	135	0	0	0	38	7	1	13	3	44	6	9	14	0.103	0.508	0.294	0.095	0.000	0.000	0.000	0.000
NAHUA 2		MEX	PUEBLA	064	41	0	0	1	16	2	0	5	0	11	2	4	0	0.000	0.609	0.256	0.000	0.085	0.000	0.000	0.049
NAHUA 3		MEX	VERA CRUZ	064	106	0	0	0	39	10	0	1	1	27	3	23	2	0.029	0.545	0.367	0.000	0.000	0.000	0.000	0.058
NASKAPI		CAN	QUEBEC	033	152	0	0	0	29	4	0	0	0	71	0	39	9	0.000	0.437	0.519	0.001	0.000	0.000	0.000	0.042
NAVAJO* 4		USA	ARIZ	063	237	0	0	3	28	18	1	2	0	85	0	67	33	0.005	0.339	0.530	0.017	0.000	0.000	0.000	0.108
PANARE*		VEN	W BOLIVAR	130	33	0	0	0	3	0	0	3	1	17	2	6	1	0.109	0.389	0.481	0.004	0.000	0.002	0.000	0.014
PARIRI*		SEE	YUPA																						
PEMON*	3P	VEN	LA SABANA	124	287	0	0	0	134	3	0	9	0	118	6	14	3	0.000	0.692	0.270	0.011	0.026	0.000	0.000	0.000
ARECUNA*		VEN	LA SABANA	124	70	0	0	0	27	0	0	4	0	33	2	2	2	0.000	0.649	0.292	0.015	0.043	0.000	0.000	0.000
CAMARACOTO*		VEN	LA SABANA	124	109	0	0	0	35	3	0	0	0	59	4	7	1	0.019	0.605	0.357	0.005	0.000	0.000	0.000	0.013
TAUREPAN*		VEN	LA SABANA	124	108	0	0	0	72	0	0	5	0	26	0	5	0	0.000	0.810	0.165	0.000	0.023	0.000	0.001	0.000
PIAROA* 1		VEN	APURE	134	110	0	0	0	16	0	0	3	0	60	0	25	6	0.011	0.431	0.526	0.028	0.003	0.000	0.000	0.000
PIRO*		PRU	R URUBANO	168	90	0	0	0	17	1	0	4	2	30	10	24	2	0.102	0.381	0.496	0.018	0.000	0.001	0.001	0.000
QUECHUA 1		ECD	NORTHERN	167	232	0	0	0	106	3	0	4	0	100	1	17	1	0.002	0.686	0.293	0.009	0.009	0.000	0.000	0.000
QUECHUA 2		ECD	CENTRAL	167	372	0	0	0	160	12	0	6	0	147	4	38	5	0.000	0.651	0.312	0.000	0.013	0.000	0.000	0.023
QUICHE 1		GUA	S CENTRAL	170	203	0	0	0	67	5	0	4	0	87	3	31	6	0.018	0.565	0.389	0.002	0.000	0.000	0.000	0.025
RAMA		NIC	RAMA CAY	175	38	0	0	0	16	1	0	0	0	15	0	5	1	0.000	0.632	0.342	0.000	0.000	0.000	0.000	0.026
SANEMA		SEE	YANOMAMA																						
SECOYA*		ECD	CUYABENDO	167	48	0	0	0	24	2	0	0	0	19	0	2	1	0.000	0.719	0.250	0.000	0.000	0.000	0.000	0.031
SHAPARU*		SEE	YUPA																						
SHIPIBO* 1		PRU	R UCAYLI	168	142	0	0	0	22	3	0	12	2	43	12	40	8	0.102	0.355	0.500	0.001	0.000	0.000	0.000	0.041
SHIPIBO* 2		PRU	R UCAYLI	043	70	0	0	0	11	3	0	13	1	17	1	15	9	0.123	0.384	0.398	0.000	0.000	0.000	0.000	0.094
SIRIONO*		BOL	R MACHUPO	169	27	0	0	0	4	0	0	0	0	12	0	11	0	0.000	0.370	0.630	0.000	0.000	0.000	0.000	0.000
SUMO*		NIC	E COAST	175	103	0	0	0	61	15	2	4	0	20	0	1	0	0.000	0.778	0.104	0.095	0.022	0.000	0.000	0.000
TAUREPAN*		SEE	PEMON																						
TERRABAS		COS	TERRABA	177	40	0	0	0	5	5	1	0	0	15	0	5	9	0.000	0.375	0.424	0.200	0.000	0.000	0.000	0.000
TICUNA*		PRU	R AMAZON	168	122	0	0	0	49	1	0	2	0	59	0	10	1	0.001	0.654	0.328	0.008	0.008	0.000	0.000	0.000
TOTONAC 2		MEX	VERA CRUZ	170	45	0	0	0	12	4	0	2	0	16	0	8	3	0.024	0.508	0.387	0.009	0.000	0.000	0.000	0.071
TOTONAC* 3		MEX	PUEBLA	064	79	0	0	0	24	6	0	4	0	30	4	9	2	0.008	0.554	0.339	0.053	0.045	0.000	0.000	0.000
TUCANO 2		BRZ	AMAZONAS	186	125	0	0	0	18	17	9	17	12	34	9	9	0	0.260	0.356	0.183	0.200	0.000	0.000	0.000	0.000
TUNEBO*		COL	COCUY MTS	131	100	0	0	0	19	0	0	0	0	31	0	45	5	0.000	0.345	0.629	0.000	0.000	0.000	0.000	0.025
TZELTAL*		MEX	CHIAPAS	170	111	0	0	0	30	7	0	0	0	47	0	21	6	0.000	0.513	0.428	0.000	0.000	0.000	0.000	0.058
TZOTZIL* 1		MEX	CHIAPAS	171	80	0	0	0	15	3	1	1	0	36	0	22	2	0.007	0.437	0.369	0.142	0.000	0.000	0.000	0.044
TZOTZIL* 2		MEX	CHIAPAS	170	91	0	0	0	22	3	0	5	0	45	1	12	3	0.014	0.530	0.400	0.034	0.021	0.000	0.000	0.000
WAICA*		SEE	YANOMAMA																						
WAPISHANA		GUY		134	119	0	0	0	32	3	0	12	0	40	0	14	18	0.000	0.495	0.356	0.093	0.055	0.000	0.000	0.000
WARAO*		VEN	ORINOCO D	134	127	0	0	0	60	3	0	2	0	46	3	12	1	0.000	0.672	0.291	0.016	0.020	0.000	0.000	0.000
XAVANTE*	3P	BRZ	MA GROSSO	091	539	0	0	2	173	34	0	27	1	226	19	52	5	0.047	0.584	0.326	0.000	0.000	0.000	0.000	0.042
YAGUA*		PRU	R AMAZON	168	9	0	0	0	5	0	0	1	0	3	0	0	0	0.000	0.777	0.063	0.000	0.056	0.000	0.104	0.000
YANOMAMA*	10P	VEN	S BOLIVAR	014	572	0	0	0	350	8	2	141	9	55	5	2	0	0.145	0.796	0.039	0.000	0.000	0.004	0.015	0.000
SANEMA*		VEN	S BOLIVAR	134	156	0	0	0	145	1	1	1	7	0	0	1	0	0.028	0.707	0.000	0.201	0.000	0.000	0.046	0.016
WAICA		VEN	S BOLIVAR	129	141	0	0	0	81	0	0	36	6	14	4	0	0	0.184	0.751	0.064	0.000	0.000	0.000	0.000	0.000
YARURO*		VEN	APURE	123	102	0	0	0	39	11	0	0	0	45	0	3	4	0.000	0.656	0.268	0.074	0.000	0.000	0.001	0.000
YUPA*	3P	VEN	E ZULIA	130	175	0	0	1	103	16	0	3	0	42	2	5	3	0.015	0.761	0.162	0.000	0.000	0.000	0.000	0.061
IRAPA* 2		VEN	E ZULIA	126	138	0	0	0	69	0	0	15	1	46	1	4	2	0.066	0.720	0.206	0.000	0.000	0.000	0.000	0.007
MACOITA* 2		VEN	E ZULIA	126	77	0	0	1	47	15	0	2	0	10	1	0	1	0.022	0.782	0.075	0.000	0.000	0.000	0.000	0.120
PARIRI*		VEN	E ZULIA	126	74	0	0	0	42	1	0	1	0	25	0	4	1	0.007	0.749	0.230	0.001	0.000	0.000	0.000	0.012
SHAPARU*		VEN	E ZULIA	126	24	0	0	0	14	0	0	0	0	7	1	1	1	0.021	0.728	0.229	0.000	0.000	0.000	0.000	0.021
ZAPOTEC		MEX	OAXACA	064	102	0	0	1	28	4	0	5	3	32	10	19	0	0.106	0.472	0.387	0.000	0.000	0.000	0.002	0.032

TABLE 3B. THE RH GENE FREQUENCIES AS DETERMINED WITH FOUR ANTI-SERA (ANTI-C, ANTI-'LITTLE C', ANTI-D, AND ANTI-E).
CONVENTIONS IN CALCULATING CHROMOSOME FREQUENCIES ARE AS DESCRIBED IN TABLE 3A.

TRIBE OR GROUP	NATION	REGION	REF	SAMPLE SIZE		PHENOTYPES												GENE FREQUENCIES							
					ANTI-C	+	+	-	+	+	-	+	+	-	+	+	-								
					ANTI-c	-	+	+	-	+	+	-	+	+	-	+	+	C	C	c	c	C	C	c	c
					ANTI-D	-	-	-	+	+	+	-	-	-	+	+	+	D	D	D	D	d	d	d	d
					ANTI-E	-	-	-	-	-	-	+	+	+	+	+	+	E	e	E	e	E	e	E	e
APACHE* 1	USA	ARIZ	042	178		0	0	0	47	22	3	0	0	0	2	40	64	0.009	0.439	0.390	0.161	0.000	0.000	0.000	0.000
CAINGANG	RRZ	R G SUL	231	166		0	0	0	64	8	3	0	0	0	11	56	24	0.051	0.592	0.287	0.069	0.000	0.000	0.000	0.000
CHAMULA*	MEX	CHIAPAS	024	57		4	0	0	19	1	1	0	0	0	7	2	23	0.068	0.283	0.345	0.101	0.000	0.202	0.000	0.000
CHIPPEWA	USA	MINN	159	161		0	0	0	16	12	0	1	0	0	2	67	63	0.000	0.313	0.357	0.200	0.129	0.000	0.000	0.000
DIEGUENO	USA	CAL	202	58		0	0	0	28	1	0	0	0	0	5	19	5	0.058	0.682	0.245	0.000	0.000	0.000	0.000	0.014
EMERILLON*	FRG		114	36		0	0	0	21	0	0	0	0	0	10	5	0	0.165	0.765	0.069	0.000	0.000	0.000	0.000	0.000
GOAJIRO* 2	VEN	W ZULIA	193	118		0	0	0	44	13	1	0	0	0	0	39	21	0.000	0.593	0.309	0.098	0.000	0.000	0.000	0.000
GUAYO*	SEE	WARAO																							
HOPI AND TEWA	USA	NM	042	106		0	0	0	20	23	21	0	0	0	0	7	35	0.000	0.330	0.233	0.436	0.000	0.000	0.000	0.000
HUASTEC* 1	MEX	VERA CRUZ	024	50		0	0	0	12	1	0	1	1	0	3	23	9	0.000	0.405	0.316	0.043	0.141	0.000	0.094	0.000
IRAPA* 1	SEE	YUPA																							
KALAPALO ANO*2	BRZ	MA GROSSO	201	73		0	0	0	22	9	0	0	0	0	4	32	6	0.050	0.586	0.281	0.000	0.000	0.000	0.000	0.082
MACOITA* 1	SEE	YUPA																							
MAM 4P	GUA	W AND SW	262	235		2	0	1	63	12	3	1	7	0	17	94	35	0.039	0.424	0.274	0.046	0.034	0.095	0.063	0.023
MARICOPA*	USA	ARIZ	042	33		0	0	0	12	3	0	0	0	0	0	8	10	0.000	0.530	0.372	0.000	0.000	0.000	0.000	0.097
MOCETENES*	ROL	AMAZONIA	223	76		0	0	0	23	8	0	0	0	0	3	22	20	0.031	0.508	0.357	0.000	0.000	0.000	0.000	0.103
NAVAJO* 2	USA	ARIZ	042	87		0	0	0	24	14	4	0	0	0	1	15	29	0.009	0.445	0.321	0.225	0.000	0.000	0.000	0.000
OAYANA*	FRG		114	100		0	0	0	37	0	0	0	0	0	6	43	14	0.047	0.598	0.355	0.000	0.000	0.000	0.000	0.000
OTOMI*	MEX	HIDALGO	024	79		0	0	0	23	5	1	1	0	0	8	24	17	0.000	0.457	0.323	0.091	0.128	0.000	0.000	0.000
OYAMPI*	FRG		114	99		0	0	0	60	0	0	0	0	0	12	24	3	0.074	0.774	0.152	0.000	0.000	0.000	0.000	0.000
PALIKOUR*	FRG		114	75		0	1	0	46	0	0	0	0	0	15	10	3	0.117	0.690	0.102	0.000	0.000	0.079	0.000	0.011
PIMA*	USA	ARIZ	042	464		0	0	0	136	59	6	0	0	0	20	139	104	0.036	0.513	0.320	0.131	0.000	0.000	0.000	0.000
POCOMAM ANO 4P	GUA	CENTRAL	262	235		2	0	1	63	12	3	1	7	0	17	94	35	0.039	0.424	0.274	0.046	0.034	0.095	0.063	0.023
RIONEGRINO*	VEN	E ZULIA	194	125		0	0	0	78	16	0	0	0	0	9	18	4	0.044	0.787	0.091	0.000	0.000	0.000	0.000	0.077
TOTONAC 1	MEX	VERA CRUZ	024	150		0	0	0	38	7	0	0	0	0	13	83	9	0.087	0.552	0.328	0.000	0.000	0.000	0.000	0.032
WARAO																									
GUAYO*	VEN	ORINOCO	120	81		0	0	0	37	3	0	0	0	0	2	37	2	0.019	0.708	0.251	0.000	0.000	0.000	0.000	0.021
WINIKINA*	VEN	ORINOCO	120	72		0	0	0	32	2	0	0	0	0	8	28	2	0.081	0.682	0.219	0.000	0.000	0.000	0.000	0.017
YARARANA*	VEN	MANAPIARE	134	14		0	0	0	3	0	0	0	0	0	2	9	0	0.153	0.524	0.234	0.000	0.000	0.000	0.088	0.000
YUMA*	USA	ARIZ	042	179		0	0	0	63	32	7	0	0	0	4	39	34	0.017	0.555	0.238	0.189	0.000	0.000	0.000	0.000
YUPA																									
IRAPA* 1	VEN	E ZULIA	193	161		0	0	0	67	8	1	0	0	0	8	70	7	0.039	0.702	0.245	0.004	0.000	0.000	0.009	0.000
MACOITA* 1	VEN	E ZULIA	195	113		0	0	0	56	28	0	0	0	0	10	19	0	0.062	0.730	0.052	0.000	0.000	0.000	0.021	0.134
RIONEGRINO*	VEN	E ZULIA	194	125		0	0	0	78	16	0	0	0	0	9	18	4	0.044	0.787	0.091	0.000	0.000	0.000	0.000	0.077

TABLE 9C. THE FREQUENCY OF THE D 'GENE' OF THE RH COMPLEX, AS DETERMINED BY AN ANTI-D SERUM. OF THE SIX SAMPLES LISTED IN THIS TABLE, IN THREE ONLY ANTI-D WAS EMPLOYED (72, 102, 108) AND IN THE REMAINING, THREE ANTISERA WERE EMPLOYED (ANTI-C, ANTI-D, ANTI-E) BUT THE CALCULATIONS LIMITED TO THE D COMPONENT.

TRIBE OR GROUP	NATION	REGION	REF	SAMPLE SIZE	PHENOTYPES RH+	RH-	GENE FREQUENCIES D+	D-
BORORO*	BRZ	MA GROSSO	197	103	0	103	0.000	1.000
CAIUA*	BRZ	MA GROSSO	197	135	0	135	0.000	1.000
CUNA* 1	PAN	S BLAS IS	072	89	0	89	0.000	1.000
GOAJIRO*	VEN	W ZULIA	118	152	0	152	0.000	1.000
NOOTKA 2	CAN	N W COAST	102	275	0	275	0.000	1.000
UTE 2	USA	UTAH	161	161	0	161	0.000	1.000

TABLE 4A. THE JK(A) AND JK(B) GENE FREQUENCIES, AS DETERMINED WITH ANTI-JK(A) AND ANTI-JK(B) SERA.

TRIBE OR GROUP	NATION	REGION	REF	SAMPLE SIZE	PHENOTYPES A+B-	A-B+	A+B+	GENE FREQUENCIES JKA	JKB	P
ACAWAIO*	GUY		130	90	22	24	44	0.489	0.511	
ARECUNA*	SEE	PEMON								
CAKCHIQUEL 1	GUA	L ALTITLN	171	141	23	47	71	0.415	0.585	
CAMARACOTO*	SEE	PEMON								
CHEROKEE *	USA	N CARLINA	209	77	17	22	38	0.468	0.532	
KEKCHI 1	GUA	COBAN	173	154	24	57	73	0.393	0.607	
LACANDON* 2	MEX	CHIAPAS	171	61	4	24	33	0.336	0.664	
MAM 1	GUA	SAN JUAN	173	112	17	62	33	0.299	0.701	A
NASKAPI	CAN	QUEBEC	033	152	41	34	77	0.523	0.477	
NAVAJO 4	USA	ARIZ	063	237	64	49	124	0.532	0.468	
PEMON* 3P	VEN	LA SABANA	124	287	57	70	160	0.477	0.523	
ARECUNA*	VEN	LA SABANA	124	70	17	23	30	0.457	0.543	
CAMARACOTO*	VEN	LA SABANA	124	109	10	26	73	0.427	0.573	B
TAUREPAN*	VEN	LA SABANA	124	108	30	21	57	0.542	0.458	
PIAROA* 1	VEN	APURE	134	109	7	43	59	0.335	0.665	
RAMA	NIC	RAMA KAY	175	36	4	8	24	0.444	0.556	
SUMO*	NIC	E COAST	175	103	22	26	55	0.481	0.519	
TAUREPAN*	SEE	PEMON								
TZOTZIL* 1	MEX	CHIAPAS	171	79	13	23	43	0.437	0.563	
WAICA*	VEN	S BOLIVAR	129	141	34	17	90	0.560	0.440	A
YARURU*	VEN	APURE	123	102	23	25	54	0.490	0.510	

TABLE 4B. THE JK(A) AND JK(B) GENE FREQUENCIES, AS DETERMINED WITH ANTI-JK(A) SERUM. (THERE IS A SINGLE STUDY (116) WITH THE USE OF ANTI-JK(B) ONLY - THIS HAS NOT BEEN TABLED).

TRIBE OR GROUP	NATION	REGION	REF	SAMPLE SIZE	PHENOTYPES		GENE FREQUENCIES	
					A+	A-	JKA	JKB
AGUARUNA*	PRU	R MARANON	168	151	67	84	0.254	0.746
ALACALUF 3	CHL	MAGELLAN	164	44	30	14	0.436	0.564
ATACAMENOS 1	CHL	ANTOFAGST	164	80	44	36	0.329	0.671
ATHABASCAN 1	USA	ALASKA	062	78	60	18	0.520	0.480
AYMARA* 1	PRU	PUNO	028	100	51	49	0.300	0.700
AYMARA* 3	PRU	PUNO	168	93	60	33	0.404	0.596
AYMARA* 4	BOL	3 AREAS P	169	503	315	188	0.389	0.611
BARI*	VEN	W ZULIA	132	70	54	16	0.522	0.478
BRIBRI*	COS	SALITRE	177	50	26	24	0.307	0.693
CABECAR*	COS	UJARRAS	177	62	45	17	0.476	0.524
CAKCHIQUEL* 2	GUA	SOLALA	170	9	9	0	1.000	0.000
CAKCHIQUEL 3	GUA	SUMPANGO	268	137	74	63	0.322	0.678
CAMPA*	PRU	R URUBMBA	168	89	67	22	0.503	0.497
CAYAPA* 2	ECD	R CAYAPAS	167	244	207	37	0.611	0.389
CHACOBO*	BOL	R YATA	169	14	11	3	0.537	0.463
CHAMA*	BOL	R MADIDI	169	30	14	16	0.270	0.730
CHOCO*	PAN	DARIEN	178	80	53	27	0.419	0.581
COLORADO* 2	ECD	S DOMINGO	167	36	24	12	0.423	0.577
CUNA* 2	PAN	S BLAS IS	178	388	256	132	0.417	0.583
GOAJIRO 3	VEN	W ZULIA	128	119	99	20	0.590	0.410
GUAYMI*	PAN	N W COAST	178	240	61	179	0.136	0.864
ICA	COL	S NEVADA	131	113	104	9	0.718	0.282
IRAPA*	SEE	YUPA						
ISCONAHUA* 2	PRU	R UCAYALI	168	14	9	5	0.402	0.598
JIVARO* 2	ECD	ARAPICOS	167	233	154	79	0.418	0.582
KEKCHI* 2	BRH	TOLEDO	176	119	73	46	0.378	0.622
LACANDON* 1	MEX	CHIAPAS	170	33	21	12	0.397	0.603
MACOITA*	SEE	YUPA						
MACUSHI*	GUY		130	119	90	29	0.506	0.494
MAKIRITARE* 1	VEN	S BOLIVAR	130	86	47	39	0.327	0.673
MAM 2	GUA	HUEHUETGO	170	24	21	3	0.646	0.354
MAM 3	GUA	W AND SW	262	70	23	47	0.181	0.819
MAPUCHE 2	CHL	LONQUIMAY	164	141	30	111	0.113	0.887
MAYA* 3	GUA	E PETEN	263	80	56	24	0.452	0.548
PANARE*	VEN	W BOLIVAR	130	33	30	3	0.699	0.301
PARIRI*	SEE	YUPA						
PIRO*	PRU	R URUBMBA	168	90	63	27	0.452	0.548
POCOMAM ANO	GUA	CENTRAL	262	40	25	15	0.388	0.612
QUECHUA 1	ECD	NORTHERN	167	228	154	74	0.430	0.570
QUECHUA 2	ECD	CENTRAL	167	372	220	152	0.361	0.639
QUICHE 1	GUA	S CENTRAL	170	203	179	24	0.656	0.344
QUICHE 2	GUA	S CENTRAL	262	80	48	32	0.367	0.633
SECOYA*	ECD	CUYABENO	167	48	29	19	0.371	0.629
SHAPARU*	SEE	YUPA						
SHIPIBO* 1	PRU	R UCAYALI	168	142	103	39	0.476	0.524
SIRIONO*	BOL	R MACHUPO	169	27	22	5	0.570	0.430
TERRABAS*	COS	TERRABA	177	40	18	22	0.258	0.742
TICUNA*	PRU	R AMAZON	168	122	91	31	0.496	0.504
TOTONAC 2	MEX	VERA CRUZ	170	45	35	10	0.529	0.471
TUCANO 2	BRZ	AMAZONAS	186	20	8	12	0.225	0.775
TUNEBO*	COL	COCUY MTS	131	98	84	14	0.622	0.378
TZELTAL*	MEX	CHIAPAS	170	111	84	27	0.507	0.493
TZOTZIL* 2	MEX	CHIAPAS	170	91	69	22	0.508	0.492
WAPISHANA	GUY		134	119	104	15	0.645	0.355
XAVANTE* 3P	BRZ	MA GROSSO	091	445	296	149	0.421	0.579

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YAGUA*            PRU   R AMAZON     168      9        7    2          0.529   0.471
YANOMAMA*  10P    VEN   S BOLIVAR    014    570      440  130          0.522   0.478
YUPA*       3P    VEN   E ZULIA      130    176      154   22          0.646   0.354
  IRAPA* 2        VEN   E ZULIA      126    138      126   12          0.705   0.295
  MACOITA* 2      VEN   E ZULIA      126     78       71    7          0.700   0.300
  PARIRI*         VEN   E ZULIA      126     74       63   11          0.614   0.386
  SHAPARU*        VEN   E ZULIA      126     24       20    4          0.592   0.408
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TABLE 5. THE FY(A) AND FY(B) GENE FREQUENCIES, AS DETERMINED BY TESTING WITH ANTI-FY(A) SERUM.

TRIBE OR GROUP	NATION	REGION	REF	SAMPLE SIZE	PHENOTYPES A+	PHENOTYPES A-	GENE FREQUENCIES FYA	GENE FREQUENCIES FYB
ACAWAIO*	GUY		130	90	76	14	0.606	0.394
AGUARUNA*	PRU	R MARANON	168	151	137	14	0.696	0.304
ALACALUF 3	CHL	MAGELLAN	164	44	38	6	0.631	0.369
APACHE 2	USA	NEW MEX	090	84	58	26	0.444	0.556
ARECUNA*	SEE	PEMON						
ATACAMENOS 1	CHL	ANTOFAGST	164	80	68	12	0.613	0.387
ATHABASCAN 1	USA	ALASKA	062	78	77	1	0.887	0.113
AWEIKOMA ANO*	BRZ	S CATRINA	233	109	78	31	0.467	0.533
AYMARA* 1	PRU	PUNO	028	58	54	4	0.737	0.263
AYMARA* 3	PRU	PUNO	168	93	82	11	0.656	0.344
AYMARA* 4	BOL	3 AREAS P	169	503	487	16	0.822	0.178
BARI*	VEN	W ZULIA	132	70	46	24	0.414	0.586
BRIBRI*	COS	SALITRE	177	50	45	5	0.684	0.316
CABECAR*	COS	UJARRAS	177	62	56	6	0.689	0.311
CAKCHIQUEL 1	GUA	L ALTITLN	171	150	135	15	0.684	0.316
CAKCHIQUEL* 2	GUA	SOLOLA	170	9	9	0	1.000	0.000
CAKCHIQUEL 3	GUA	SUMPANGO	268	137	125	12	0.704	0.296
CAMARACOTO*	SEE	PEMON						
CAMPA*	PRU	R URUBAMB	168	89	86	3	0.816	0.184
CARAJA*	BRZ	S ISABEL	108	40	27	13	0.430	0.570
CAYAPA* 2	ECD	R CAYAPAS	167	244	229	15	0.752	0.248
CHACOBO*	BOL	R YALTA	169	14	12	2	0.622	0.378
CHAMA*	BOL	R MADIDI	169	30	30	0	1.000	0.000
CHEROKEE*	USA	N CARLINA	209	78	62	16	0.547	0.453
CHINANTEC	MEX	OAXACA	064	20	17	3	0.613	0.387
CHIPPEWA	USA	MINN	159	161	158	3	0.864	0.136
CHOCO*	PAN	DARIEN	178	80	71	9	0.665	0.335
CHOL	MEX	CHIAPAS	064	137	126	11	0.717	0.283
CHONTOL	MEX	TABASCO	064	101	90	11	0.670	0.330
COLORADO* 2	ECD	S DOMINGO	167	36	29	7	0.559	0.441
CORA*	MEX	NAYARIT	064	96	95	1	0.898	0.102
CUIVA*	VEN	APURE	134	82	72	10	0.651	0.349
CUNA* 2	PAN	S BLAS IS	178	388	343	45	0.659	0.341
DIEGUENO	USA	CAL	202	58	52	6	0.678	0.322
EMERILLON*	FRG		114	27	15	12	0.333	0.667
GOAJIRO 3	VEN	W ZULIA	128	119	106	13	0.669	0.331
GUAHIBO*	VEN	APURE	134	109	102	7	0.747	0.253
GUARANI* 2	BRZ	S CATRINA	233	33	23	10	0.450	0.550
GUAYMI*	PAN	N W COAST	178	240	183	57	0.513	0.487
GUAYO*	SEE	WARAO						
HUASTEC 2	MEX	VERA CRUZ	064	75	67	8	0.673	0.327
HUICHOL	MEX	JALISCO	064	69	62	7	0.681	0.319
ICA	COL	S NEVADA	131	113	107	6	0.770	0.230
IRAPA* 1	SEE	YUPA						
IRAPA* 2	SEE	YUPA						
ISCONAHUA* 1	PRU	R UCAYALI	043	16	10	6	0.388	0.612
ISCONAHUA* 2	PRU	R UCAYALI	168	14	6	8	0.244	0.756
JIVARO* 2	ECD	ARAPICOS	167	233	192	41	0.580	0.420
KALAPALO ANO*2	BRZ	MA GROSSO	201	73	0	73	0.000	1.000
KEKCHI 1	GUA	COBAN	173	162	138	24	0.615	0.385
KEKCHI* 2	BRH	TOLEDO	176	119	112	7	0.757	0.243
LACANDON* 1	MEX	CHIAPAS	170	33	32	1	0.826	0.174
LACANDON* 2	MEX	CHIAPAS	171	60	57	3	0.776	0.224
MACOITA* 1	SEE	YUPA						
MACOITA* 2	SEE	YUPA						
MACUSHI*	GUY		130	119	108	11	0.696	0.304
MAKIRITARE* 1	VEN	S BOLIVAR	130	86	83	3	0.813	0.187

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MAM 1               GUA  SAN JUAN      171   116     110    6      0.773  0.227
MAM 2               GUA  HUEHUETGO     170    24      24    0      1.000  0.000
MAM 3               GUA  W AND SW      262    70      52   18      0.493  0.507
MAPUCHE 2           CHL  LONQUIMAY     164   141     129   12      0.708  0.292
MAYA* 3             GUA  E PETEN       263   120      98   22      0.572  0.428
MAZATEC 1           MEX  OAXACA        064   136     103   33      0.507  0.493
NAHUA 1             MEX  PUEBLA        064    41      40    1      0.844  0.156
NAHUA 3             MEX  VERA CRUZ     064   141     123   18      0.643  0.357
NASKAPI             CAN  QUEBEC        033   152     149    3      0.860  0.140
NAVAJO 4            USA  ARIZ          063   237     219   18      0.724  0.276
OAYANA*             FRG                114    71      64    7      0.686  0.314
OYAMPI*             FRG                114    22      14    8      0.397  0.603
PALIKOUR*           FRG                114    74      50   24      0.431  0.569
PANARE*             VEN  W BOLIVAR     130    33      30    3      0.699  0.301
PARIRI*             SEE   YUPA
PEMON*        3P    VEN  LA SABANA     124   287     266   21      0.729  0.271
  ARECUNA*          VEN  LA SABANA     124    70      65    5      0.733  0.267
  CAMARACOTO*       VEN  LA SABANA     124   109      96   13      0.655  0.345
  TAUREPAN*         VEN  LA SABANA     124   108     105    3      0.833  0.167
PIAROA* 1           VEN  APURE         134    81      60   21      0.491  0.509
PIMA*               USA  ARIZ          042   184     179    5      0.835  0.165
PIRO*               PRU  R URUBAMB     168    90      76   14      0.606  0.394
POCOMAM             GUA  CENTRAL       062    80      63   17      0.539  0.461
QUECHUA 1           ECD  NORTHERN      167   232     220   12      0.773  0.227
QUECHUA 2           ECD  SOUTHERN      167   372     344   28      0.726  0.274
QUICHE 1            GUA  S CENTRAL     170   203     183   20      0.686  0.314
QUICHE 2            GUA  S CENTRAL     262    80      65   15      0.567  0.433
RAMA                NIC  RAMA CAY      175    37      33    4      0.671  0.329
SANEMA*             SEE   YANOMAMA
SECOYA*             ECD  CUYABENDO     167    48      41    7      0.618  0.382
SHAPARU*            SEE   YUPA
SHIPIBO* 1          PRU  R UCAYALI     168   142     138    4      0.832  0.168
SHIPIBO* 2          PRU  R UCAYALI     043    70      66    4      0.761  0.239
SIRIONO*            BOL  R MACHUPO     169    27      27    0      1.000  0.000
SUMO*               NIC  E COAST       175   103      98    5      0.780  0.220
TAUREPAN*           SEE   PEMON
TERRABAS*           COS  TERRABA       177    40      37    3      0.726  0.274
TICUNA*             PRU  R AMAZON      168   122     104   18      0.616  0.384
TOTONAC 2           MEX  VERA CRUZ     170    45      43    2      0.789  0.211
TOTONAC 3           MEX  PUEBLA        064    79      79    0      1.000  0.000
TUCANO 2            BRZ  AMAZONAS      186    20       8   12      0.225  0.775
TUNEBO*             COL  COCUY MTS     131   100      90   10      0.684  0.316
TZELTAL*            MEX  CHIAPAS       170   111      94   17      0.609  0.391
TZOTZIL* 1          MEX  CHIAPAS       171    80      76    4      0.776  0.224
TZOTZIL* 2          MEX  CHIAPAS       170    91      83    8      0.704  0.296
WAICA*              SEE   YANOMAMA
WAPISHANA           GUY                134   119     102   17      0.622  0.378
WARAO*              VEN  ORINOCO D     134   127     115   12      0.693  0.307
  GUAYO*            VEN  ORINOCO       120    81      74    7      0.706  0.294
  WINIKINA*         VEN  ORINOCO       120    72      70    2      0.833  0.167
XAVANTE*      3P    BRZ  MA GROSSO     091   539     425  114      0.540  0.460
YABARANA*           VEN  MANAPIARE     134    14      11    3      0.537  0.463
YAGUA*              PRU  R AMAZON      168     9       9    0      1.000  0.000
YANOMAMA*    10P    VEN  S BOLIVAR     014   491     434   57      0.659  0.341
  SANEMA*           VEN  S BOLIVAR     134   156     128   28      0.576  0.424
  WAICA*            VEN  S BOLIVAR     129   141     117   24      0.587  0.413
YARURO*             VEN  APURE         123   102      79   23      0.525  0.475
YUPA          3P    VEN  E ZULIA       130   176     133   43      0.506  0.494
  IRAPA* 1          VEN  E ZULIA       193   154      97   57      0.392  0.608
  IRAPA* 2          VEN  E ZULIA       126   138     112   26      0.566  0.434

  MACOITA* 1        VEN  E ZULIA       195   117      63   54      0.321  0.679
  MACOITA* 2        VEN  E ZULIA       126    78      53   25      0.434  0.566
  PARIRI*           VEN  E ZULIA       126    74      62   12      0.597  0.403
  SHAPARU*          VEN  E ZULIA       126    24      18    6      0.500  0.500
ZAPOTEC             MEX  OAXACA        064   105      82   23      0.532  0.468
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TABLF 6. THE DI(A) AND DI(B) GENF FREQUENCIES. AS DETERMINED BY TESTING WITH AN ANTI-DI(A) SERUM.

TRIBE OR GROUP	NATION	REGION	REF	SAMPLE SIZE	PHENOTYPES DIA+	DIA-	GENE FREQUENCIES DIA	DIB
ACAWAIO*	GUY		130	90	37	53	0.233	0.767
AGUARUNA*	PRU	R MARANON	168	151	12	139	0.041	0.959
ALACALUF* 3	CHL	MAGELLAN	164	44	0	44	0.000	1.000
APACHE 2	USA	NEW MEX	090	108	4	104	0.019	0.981
ARECUNA*	SEE	PEMON						
ATACAMENOS 1	CHL	ANTOFAGST	164	80	7	73	0.045	0.955
ATHABASCAN 1	USA	ALASKA	062	78	0	78	0.000	1.000
AWEIKOMA ANO*	BRZ	S CATRINA	233	109	59	50	0.323	0.677
AYMARA* 1	PRU	PUNO	028	58	10	48	0.090	0.910
AYMARA* 2	BOL	MOCOMOCO	259	100	4	96	0.020	0.980
AYMARA* 3	PRU	PUNO	168	93	15	78	0.084	0.916
AYMARA* 4	BOL	3 AREAS P	169	491	48	443	0.050	0.950
BARI*	VEN	W ZULIA	132	126	0	126	0.000	1.000
BRIBRI*	COS	SALITRE	177	50	0	50	0.000	1.000
CARECAR*	COS	UJARRAS	177	62	0	62	0.000	1.000
CAINGANG	BRZ	R G SUL	231	257	72	185	0.152	0.848
CAKCHIQUEL 1	GUA	L ALTITLN	171	150	13	137	0.044	0.956
CAKCHIQUEL* 2	GUA	SOLOLA	170	5	0	5	0.000	1.000
CAMARACOTO*	VEN	LA SABANA	124	109	19	90	0.091	0.909
CAMPA*	PRU	R URUBAMB	168	89	38	51	0.243	0.757
CARAJA*	BRZ	S ISABEL	108	36	13	23	0.201	0.799
CARINA*	VEN	MONAGAS	118	170	50	120	0.160	0.840
CAYAPA* 2	ECD	R CAYAPAS	167	240	21	219	0.045	0.955
CHACOBO*	BOL	R YATA	169	14	4	10	0.155	0.845
CHAMA*	BOL	R MADIDI	169	30	22	8	0.484	0.516
CHEROKEE*	USA	N CARLINA	209	78	0	78	0.000	1.000
CHINANTEC	MEX	OAXACA	064	20	4	16	0.105	0.895
CHIPPEWA	USA	MINN	143	148	16	132	0.056	0.944
CHOCO*	PAN	DARIEN	178	80	53	27	0.419	0.581
CHOL	MEX	CHIAPAS	064	54	7	47	0.067	0.933
CHONTOL	MEX	TABASCO	064	89	17	72	0.101	0.899
COLORADO* 2	ECD	S DOMINGO	167	36	1	35	0.014	0.986
CORA*	MEX	NAYARIT	064	96	23	73	0.128	0.872
CUIVA*	VEN	APURE	134	82	0	82	0.000	1.000
CUNA* 2	PAN	S BLAS IS	178	317	24	293	0.039	0.961
EMERILLON*	FRG		114	28	4	24	0.074	0.926
GOAJIRO* 1	VEN	W ZULIA	118	152	8	144	0.027	0.973
GOAJIRO 3	VEN	W ZULIA	128	119	12	107	0.052	0.948
GUAHIBO* 1	VEN	APURE	134	109	23	86	0.112	0.888
GUAHIBO* 2	VEN	APURE	118	76	11	65	0.075	0.925
GUARANI*	BRZ	S CATRINA	233	34	14	20	0.233	0.767
GUAYMI*	PAN	N W COAST	178	240	1	239	0.002	0.998
GUAYO*	SEE	WARAO						
HUASTEC 2	MEX	VERA CRUZ	064	70	5	65	0.036	0.964
HUICHOL	MEX	JALISCO	064	72	29	43	0.227	0.773
ICA	COL	S NEVADA	131	112	47	65	0.238	0.762
IRAPA* 1 AND 2	SEE	YUPA						
ISCONAHUA* 1	PRU	R UCAYALI	043	16	4	12	0.134	0.866
ISCONAHUA* 2	PRU	R UCAYALI	168	14	4	10	0.155	0.845
JIVARO* 2	ECD	ARAPICOS	167	233	37	196	0.083	0.917
KEKCHI 1	GUA	CORAN	173	122	7	115	0.029	0.971
KEKCHI* 2	BRH	TOLEDO	176	117	8	109	0.035	0.965
LACANDON* 1	MEX	CHIAPAS	170	33	11	22	0.184	0.816
LACANDON* 2	MEX	CHIAPAS	171	61	10	51	0.086	0.914
MACOITA* 1 2	SEE	YUPA						
MACUSHI*	GUY		130	119	34	85	0.155	0.845
MAKIRITARE* 1	VEN	S BOLIVAR	130	86	26	60	0.165	0.835
MAM 1	GUA	SAN JUAN	171	116	12	104	0.053	0.947

MAM 2		GUA	W AND SW	262	70	23	47	0.181	0.819
MAPUCHE 2		CHL	LONQUIMAY	164	130	5	125	0.019	0.981
MAYA* 3		GUA	E PETEN	263	120	36	84	0.163	0.837
MAZATEC 1		MEX	OAXACA	064	136	16	120	0.061	0.939
NAHUA 1		MEX	PUEBLA	064	32	2	30	0.032	0.968
NAHUA 3		MEX	VERA CRUZ	064	128	18	110	0.073	0.927
NASKAPI		CAN	QUEBEC	033	152	13	139	0.044	0.956
NAVAJO 4		USA	ARIZ	063	237	11	226	0.023	0.977
OAYANA*		FRG		114	91	17	74	0.098	0.902
OYAMPI*		FRG		114	98	34	64	0.192	0.808
PALIKOUR*		FRG		114	30	11	19	0.204	0.796
PANARE*		VEN	W BOLIVAR	130	33	19	14	0.349	0.651
PARIRI*		SEE	YUPA						
PEMON*	3P	VEN	LA SABANA	124	287	78	209	0.147	0.853
ARECUNA*		VEN	LA SABANA	124	70	25	45	0.198	0.802
CAMARACOTO*		VEN	LA SABANA	124	109	19	90	0.091	0.909
TAUREPAN*		VEN	LA SABANA	124	108	34	74	0.172	0.828
PIAROA* 1		VEN	APURE	134	111	19	92	0.090	0.910
PIAROA* 2		VEN	AMAZONAS	134	24	3	21	0.065	0.935
PIRO*		PRU	R URUBAMB	168	90	33	57	0.204	0.796
POCOMAM ANO		GUA	CENTRAL	262	80	17	63	0.113	0.887
QUECHUA 1		ECD	NORTHERN	167	228	50	178	0.116	0.884
QUECHUA 2		ECD	SOUTHERN	167	355	92	263	0.139	0.861
QUICHE 1		GUA	S CENTRAL	170	46	8	38	0.091	0.909
QUICHE 2		GUA	S CENTRAL	262	80	25	55	0.171	0.829
RAMA		NIC	RAMA CAY	175	37	0	37	0.000	1.000
RIONEGRINO*		SEE	YUPA						
SANEMA*		SEE	YANOMAMA						
SECOYA*		ECD	CUYARENDO	167	48	9	39	0.099	0.901
SHAPARU*		SEE	YUPA						
SHIPIRO* 1		PRU	R UCAYALI	168	141	91	50	0.405	0.595
SHIPIRO* 2		PRU	R UCAYALI	043	70	42	28	0.367	0.633
SIRIONO*		BOL	R MACHURO	169	27	1	26	0.019	0.981
SUMO*		NIC	E COAST	175	103	11	92	0.055	0.945
TAUREPAN*		SEE	PEMON						
TERRABAS*		COS	TERRABA	177	40	0	40	0.000	1.000
TICUNA*		PRU	R AMAZON	168	122	44	78	0.200	0.800
TOTONAC 2		MEX	VERA CRUZ	170	43	9	34	0.111	0.889
TOTONAC 3		MEX	PUEBLA	064	49	4	45	0.042	0.958
TUCANO 2		BRZ	AMAZONAS	186	131	46	85	0.194	0.806
TUNERO*		COL	COCUY MTS	131	100	1	99	0.005	0.995
TZELTAL*		MEX	CHIAPAS	170	111	11	100	0.051	0.949
TZOTZIL*	1	MEX	CHIAPAS	171	80	15	65	0.099	0.901
TZOTZIL* 2		MEX	CHIAPAS	170	86	13	73	0.079	0.921
WAICA*		SEE	YANOMAMA						
WAPISHANA		GUY		134	119	33	86	0.150	0.850
WARAO*		VEN	ORINOCO D	134	127	0	127	0.000	1.000
GUAYO*		VEN	ORINOCO	120	81	3	78	0.019	0.981
WINIKINA*		VEN	ORINOCO	120	72	0	72	0.000	1.000
XAVANTE*	3P	BRZ	MA GROSSO	091	538	164	374	0.166	0.834
YABARANA*		VEN	MANAPIARE	134	14	9	5	0.402	0.598
YAGUA*		PRU	R AMAZON	168	9	2	7	0.118	0.882
YANOMAMA*	10P	VEN	S BOLIVAR	014	571	0	571	0.000	1.000
SANEMA*		VEN	S BOLIVAR	134	156	9	147	0.029	0.971
WAICA*		VEN	S BOLIVAR	129	141	0	141	0.000	1.000
YARURO*		VEN	APURE	123	102	5	97	0.025	0.975
YUPA*	3P	VEN	E ZULIA	130	176	46	130	0.141	0.859
IRAPA* 1		VEN	E ZULIA	193	44	0	44	0.000	1.000
IRAPA* 2		VEN	E ZULIA	121	138	3	135	0.011	0.989
MACOITA* 1		VEN	E ZULIA	195	117	24	93	0.108	0.892
MACOITA* 2		VEN	E ZULIA	126	78	17	61	0.116	0.884
PARIRI*		VEN	E ZULIA	126	74	25	49	0.186	0.814
RIONEGRINO*		VEN	E ZULIA	194	125	31	94	0.133	0.867
SHAPARU*		VEN	E ZULIA	126	24	6	18	0.134	0.866
ZAPOTEC		MEX	OAXACA	064	105	15	90	0.074	0.926

TABLE 7. THE P GENE FREQUENCIES. AS DETERMINED BY TESTING WITH AN ANTI-P SERUM. FOR 'PP' READ CAPITAL P AND FOR 'P', LOWER CASE P.

TRIBE OR GROUP	NATION	REGION	REF	SAMPLE SIZE	PHENOTYPES P1	PHENOTYPES P2	GENE FREQUENCIES PP	GENE FREQUENCIES P
ACAWAIO*	GUY		130	90	64	26	0.463	0.537
AGUARUNA*	PRU	R MARANON	168	151	140	11	0.730	0.270
ALACALUF 3	CHL	MAGELLAN	164	44	25	19	0.343	0.657
APACHE 2	USA	NEW MEX	090	61	35	26	0.347	0.653
ARECUNA*	SEE	PEMON						
ATACAMENOS	CHL	ANTOFAGST	164	80	61	19	0.513	0.487
ATHABASCAN 1	USA	ALASKA	062	77	14	63	0.095	0.905
AWEIKOMA ANO*	BRZ	S CATRINA	233	122	41	81	0.185	0.815
AYMARA* 1	PRU	PUNO	028	58	21	37	0.201	0.799
AYMARA* 3	PRU	PUNO	168	93	92	1	0.896	0.104
AYMARA* 4	BOL	3 AREAS P	169	503	434	69	0.630	0.370
BARI*	VEN	W ZULIA	132	70	32	38	0.263	0.737
BRIBRI*	COS	SALITRE	177	50	42	8	0.600	0.400
CABECAR*	COS	UJARRAS	177	62	61	1	0.873	0.127
CAINGANG	BRZ	R G SUL	231	166	73	93	0.252	0.748
CAKCHIQUEL 1	GUA	L ALTITLN	171	150	116	34	0.524	0.476
CAKCHIQUEL 3	GUA	SUMPANGO	268	137	85	52	0.384	0.616
CAMARACOTO*	SEE	PEMON						
CAMPA*	PRU	R URUBMBA	168	89	66	23	0.492	0.508
CAYAPA* 2	ECD	R CAYAPAS	167	244	134	110	0.329	0.671
CHACOBO*	BOL	R YATA	169	14	14	0	1.000	0.000
CHAMA*	BOL	R MADIDI	169	30	20	10	0.423	0.577
CHEROKEE*	USA	N CARLINA	209	78	65	13	0.592	0.408
CHINANTEC	MEX	OAXACA	064	20	8	12	0.225	0.775
CHIPPEWA	USA	MINN	159	161	136	25	0.606	0.394
CHOCO*	PAN	DARIEN	178	80	63	17	0.539	0.461
CHOL	MEX	CHIAPAS	064	150	47	103	0.171	0.829
CHONTOL	MEX	TABASCO	064	101	55	46	0.325	0.675
COLORADO* 2	ECD	S DOMINGO	167	36	29	7	0.559	0.441
CORA*	MEX	NAYARIT	064	96	74	22	0.521	0.479
CUIVA*	VEN	APURE	134	82	64	18	0.531	0.469
CUNA* 2	PAN	S BLAS IS	178	388	328	60	0.607	0.393
DIEGUENO	USA	CAL	202	58	33	25	0.343	0.657
EMERILLON*	FRG		114	7	3	4	0.244	0.756
GOAJIRO 3	VEN	W ZULIA	128	119	63	56	0.314	0.686
GUAHIBO*	VEN	APURE	134	109	67	42	0.379	0.621
GUARANI* 2	BRZ	S CATRINA	233	34	24	10	0.458	0.542
GUAYMI*	PAN	N W COAST	178	240	197	43	0.577	0.423
HUASTEC 2	MEX	VERA CRUZ	064	75	56	19	0.497	0.503
HUICHOL	MEX	JALISCO	064	71	67	4	0.763	0.237
ICA	COL	S NEVADA	131	113	41	72	0.202	0.798
IRAPA*	SEE	YUPA						
ISCONAHUA* 2	PRU	R UCAYALI	168	14	14	0	1.000	0.000
JIVARO* 2	ECD	ARAPICOS	167	233	183	50	0.537	0.463
KALAPALO ANO*2	BRZ	MA GROSSO	201	73	30	43	0.233	0.767
KEKCHI 1	GUA	COBAN	173	162	137	25	0.607	0.393
KEKCHI* 2	BRH	TOLEDO	176	119	106	13	0.669	0.331
LACANDON* 1	MEX	CHIAPAS	170	33	24	9	0.478	0.522
LACANDON* 2	MEX	CHIAPAS	171	61	47	14	0.521	0.479
MACOITA*	SEE	YUPA						
MACUSHI*	GUY		130	119	79	40	0.420	0.580
MAKIRITARE* 1	VEN	S BOLIVAR	130	86	57	29	0.419	0.581
MAM 1	GUA	SAN JUAN	171	116	89	27	0.518	0.482
MAM 3	GUA	W AND SW	262	70	38	32	0.324	0.676
MAPUCHE 2	CHL	LONQUIMAY	164	141	137	4	0.832	0.168

MAYA* 3		GUA	E PETEN	263	120	85	35	0.460	0.540
MAZATEC 1		MEX	OAXACA	064	136	43	93	0.173	0.827
NAHUA 1		MEX	PUEBLA	064	41	23	18	0.337	0.663
NAHUA 3		MEX	VERA CRUZ	064	141	102	39	0.474	0.526
NASKAPI		CAN	QUEBEC	033	152	108	44	0.462	0.538
NAVAJO 4		USA	ARIZ	063	237	142	95	0.367	0.633
OYANA*		FRG		114	99	46	53	0.268	0.732
PALIKOUR*		FRG		114	75	38	37	0.298	0.702
PANARE*		VEN	W BOLIVAR	130	33	8	25	0.130	0.870
PARIRI*		SEE	YUPA						
PEMON*	3P	VEN	LA SABANA	124	287	149	138	0.307	0.693
ARECUNA*		VEN	LA SABANA	124	70	36	34	0.303	0.697
CAMARACOTO*		VEN	LA SABANA	124	109	54	55	0.290	0.710
TAUREPAN*		VEN	LA SABANA	124	108	59	49	0.326	0.674
PIAROA* 1		VEN	APURE	134	111	84	27	0.507	0.493
PIRO*		PRU	R URUBMBA	168	90	75	15	0.592	0.408
POCOMAM ANO		GUA	CENTRAL	262	80	59	21	0.488	0.512
QUECHUA 1		ECD	NORTHERN	167	221	204	17	0.723	0.277
QUECHUA 2		ECD	CENTRAL	167	372	338	34	0.698	0.302
QUICHE 2		GUA	S CENTRAL	262	80	55	25	0.441	0.559
RAMA		NIC	RAMA KAY	175	37	31	6	0.597	0.403
SANEMA*		SEE	YANOMAMA						
SECOYA*		ECD	CUYABENO	167	48	29	19	0.371	0.629
SHAPARU*		SEE	YUPA						
SHIPIBO* 1		PRU	R UCAYALI	168	142	109	33	0.518	0.482
SIRIONO*		BOL	R MACHUPO	169	27	20	7	0.491	0.509
SUMO*		NIC	E COAST	175	103	64	39	0.385	0.615
TAUREPAN*		SEE	PEMON						
TERRABAS*		COS	TERRABA	177	40	38	2	0.776	0.224
TICUNA*		PRU	R AMAZON	168	122	121	1	0.909	0.091
TOTONAC 2		MEX	VERA CRUZ	170	45	27	18	0.367	0.633
TOTONAC 3		MEX	PUEBLA	064	79	50	29	0.394	0.606
TUNEBO*		COL	COCUY MTS	131	100	54	46	0.322	0.678
TZELTAL*		MEX	CHIAPAS	170	101	68	33	0.428	0.572
TZOTZIL* 1		MEX	CHIAPAS	171	80	67	13	0.597	0.403
TZOTZIL* 2		MEX	CHIAPAS	170	75	54	21	0.471	0.529
WAICA*		SEE	YANOMAMA						
WARAO*		VEN	ORINOCO D	134	127	39	88	0.168	0.832
XAVANTE*	3P	BRZ	MA GROSSO	091	538	468	70	0.639	0.361
YAGUA*		PRU	R AMAZON	168	9	9	0	1.000	0.000
YANOMAMA*	10P	VEN	S BOLIVAR	014	570	427	143	0.499	0.501
SANEMA*		VEN	S BOLIVAR	134	156	79	77	0.297	0.703
WAICA*		VEN	S BOLIVAR	129	141	81	60	0.348	0.652
YARURO*		VEN	APURE	123	102	62	40	0.374	0.626
YUPA*	3P	VEN	E ZULIA	130	176	76	100	0.246	0.754
IRAPA*		VEN	E ZULIA	126	138	74	64	0.319	0.681
MACOITA*		VEN	E ZULIA	126	78	44	34	0.340	0.660
PARIRI*		VEN	E ZULIA	126	74	23	51	0.170	0.830
SHAPARU*		VEN	E ZULIA	126	24	9	15	0.209	0.791
ZAPOTEC		MEX	OAXACA	064	105	41	64	0.219	0.781

TABLE 8A. THE PHENOTYPE FREQUENCIES FOR THE LEWIS SYSTEM, AS DETERMINED BY TESTING WITH ANTI-LE(A) AND ANTI-LE(B) SERA. GENE FREQUENCIES CANNOT BE ROUTINELY COMPUTED BECAUSE THE ABH SECRETOR STATUS WHICH IS NECESSARY TO SUCH CALCULATIONS WAS NOT DETERMINED FOR MOST SAMPLES.

TRIBE OR GROUP	NATION	REGION	REF	SAMPLE SIZE	PHENOTYPES			PHENOTYPE FREQUENCIES		
					A-B-	A+B-	A-B+	A-B-	A+B-	A-B+
AGUARUNA*	PRU	R MARANON	168	151	28	0	123	0.185	0.000	0.815
ALACALUF* 3	CHL	MAGELLAN	164	44	10	0	34	0.227	0.000	0.773
APACHE 2	USA	NEW MEX	090	49	10	0	39	0.204	0.000	0.796
ATHABASCAN 1	USA	ALASKA	062	77	4	0	73	0.052	0.000	0.948
AYMARA* 3	PRU	PUNO	168	93	4	0	89	0.043	0.000	0.957
AYMARA* 4	BOL	3 AREAS P	169	503	48	2	453	0.095	0.004	0.900
BRIBRI*	COS	SALITRE	177	50	16	0	34	0.320	0.000	0.680
CABECAR*	COS	UJARRAS	177	62	20	0	42	0.323	0.000	0.677
CAKCHIQUEL* 1	GUA	L ALTITLN	171	150	27	1	122	0.180	0.007	0.812
CAKCHIQUEL* 2	GUA	SOLALA	170	9	4	0	5	0.444	0.000	0.556
CAMPA*	PRU	R URUBMBA	168	89	32	0	57	0.360	0.000	0.640
CHACOBO*	BOL	R YATA	169	14	0	0	14	0.000	0.000	1.000
CHAMA*	BOL	R MADIDI	169	30	11	0	19	0.367	0.000	0.633
CHOCO*	PAN	DARIEN	178	80	27	18	35	0.337	0.225	0.437
COLORADO* 2	ECD	S DOMINGO	167	36	11	0	25	0.306	0.000	0.694
CUNA* 2	PAN	S BLAS IS	178	388	173	0	215	0.446	0.000	0.554
GUAYMI*	PAN	N W COAST	178	240	123	0	117	0.512	0.000	0.488
ISCONAHUA* 2	PRU	R UCAYALI	168	14	3	0	11	0.214	0.000	0.786
JIVARO* 2	ECD	ARAPICOS	167	233	106	0	127	0.455	0.000	0.545
KEKCHI 1	GUA	COBAN	173	162	32	0	130	0.198	0.000	0.802
KEKCHI* 2	BRH	TOLEDO	176	119	91	0	28	0.765	0.000	0.235
LACANDON* 1	MEX	CHIAPAS	170	33	10	0	23	0.303	0.000	0.697
LACANDON* 2	MEX	CHIAPAS	171	61	9	0	52	0.148	0.000	0.852
MAM 1	GUA	SAN JUAN	171	116	34	0	82	0.293	0.000	0.707
MAM 2	GUA	HUEHUETGO	170	24	5	1	18	0.208	0.042	0.750
MAPUCHE 2	CHL	LONQUIMAY	164	141	18	0	123	0.128	0.000	0.872
NASKAPI	CAN	QUEBEC	033	152	25	0	127	0.164	0.000	0.836
NAVAJO 4	USA	ARIZ	063	237	24	1	212	0.101	0.004	0.895
PIRO*	PRU	R URUBMBA	168	90	17	0	73	0.189	0.000	0.811
QUICHE 1	GUA	S CENTRAL	170	203	42	2	159	0.207	0.010	0.782
QUECHUA 1	ECD	NORTHERN	167	231	73	0	158	0.316	0.000	0.684
QUECHUA 2	ECD	CENTRAL	167	368	110	0	258	0.299	0.000	0.701
RAMA	NIC	RAMA KAY	175	37	21	0	16	0.568	0.000	0.432
SHIPIBO* 1	PRU	R UCAYALI	168	142	29	0	113	0.204	0.000	0.796
SIRIONO*	BOL	R MACHUPO	169	27	4	0	23	0.148	0.000	0.852
SUMO*	NIC	E COAST	175	103	37	13	53	0.359	0.126	0.515
TERRABAS*	COS	TERRABA	177	39	16	0	23	0.410	0.000	0.590
TICUNA*	PRU	R AMAZON	168	122	2	0	120	0.016	0.000	0.984
TOTONAC 2	MEX	VERA CRUZ	170	45	8	0	37	0.178	0.000	0.822
TZELTAL*	MEX	CHIAPAS	170	111	26	1	84	0.234	0.009	0.757
TZOTZIL* 1	MEX	CHIAPAS	171	80	13	0	67	0.163	0.000	0.837
TZOTZIL* 2	MEX	CHIAPAS	170	91	16	2	73	0.176	0.022	0.801
XAVANTE* 3P	BRZ	MA GROSSO	091	338	64	102	172	0.189	0.302	0.508
YAGUA*	PRU	R AMAZON	168	9	0	0	9	0.000	0.000	1.000

TABLE 8B. THE PHENOTYPE FREQUENCIES FOR THE LEWIS SYSTEM. AS DETERMINED BY TESTING WITH ANTI-LE(A). GENE FREQUENCIES CANNOT BE ROUTINELY COMPUTED BECAUSE THE ABH SECRETOR STATUS WHICH IS NECESSARY TO SUCH CALCULATIONS WAS NOT DETERMINED FOR MOST SAMPLES.

TRIBE OR GROUP	NATION	REGION	REF	SAMPLE SIZE	PHENOTYPES A+	PHENOTYPES A-	PHENOTYPE FREQUENCIES A+	PHENOTYPE FREQUENCIES A-
ACAWAIO*	GUY		130	90	0	90	0.000	1.000
ARECUNA*	SEE	PEMON						
ATACAMENOS 1	CHL	ANTOFAGST	164	80	2	78	0.025	0.975
AWEIKOMA ANO	BRZ	S CATRINA	233	118	69	49	0.585	0.415
AYMARA* 1	PRU	PUNO	028	58	0	58	0.000	1.000
BARI*	VEN	W ZULIA	132	70	0	70	0.000	1.000
CAMARACOTO*	SEE	PEMON						
CUIVA*	VEN	APURE	134	82	0	82	0.000	1.000
GOAJIRO 3	VEN	W ZULIA	128	119	0	119	0.000	1.000
GUAHIBO*	VEN	APURE	134	109	1	108	0.009	0.991
GUARANI* 2	VEN	S CATRINA	233	34	22	12	0.647	0.353
ICA	COL	S NEVADA	131	113	0	113	0.000	1.000
IRAPA*	SEE	YUPA						
ISCONAHUA* 1	PRU	R UCAYALI	043	16	0	16	0.000	1.000
MACOITA*								
MACUSHI*	GUY		130	119	0	119	0.000	1.000
MAKIRITARE* 1	VEN	S BOLIVAR	130	86	0	86	0.000	1.000
OTOMI*	MEX	HIDALGO	024	81	8	73	0.099	0.901
PANARE*	VEN	W BOLIVAR	130	33	0	33	0.000	1.000
PARIRI*	SEE	YUPA						
PEMON* 3P	VEN	LA SABANA	124	287	1	286	0.003	0.997
ARECUNA*	VEN	LA SABANA	124	70	0	70	0.000	1.000
CAMARACOTO*	VEN	LA SABANA	124	109	1	108	0.009	0.991
TAUREPAN*	VEN	LA SABANA	124	108	0	108	0.000	1.000
PIAROA* 1	VEN	APURE	134	81	0	81	0.000	1.000
SANEMA*	SEE	YANOMAMA						
SECOYA*	ECD	CUYABENO	167	48	0	48	0.000	1.000
SHAPARU*	SEE	YUPA						
SHIPIBO* 2	PRU	R UCAYALI	043	70	0	70	0.000	1.000
TAUREPAN*	SEE	PEMON						
TUNEBO*	COL	COCUY MTS	131	99	0	99	0.000	1.000
WAICA*	SEE	YANOMAMA						
WARAO*	VEN	ORINOCO D	134	127	0	127	0.000	1.000
YANOMAMA								
SANEMA*	VEN	S BOLIVAR	134	156	0	156	0.000	1.000
WAICA*	VEN	S BOLIVAR	129	141	0	141	0.000	1.000
YARURO*	VEN	APURE	123	102	0	102	0.000	1.000
YUPA* 3P	VEN	E ZULIA	130	176	0	176	0.000	1.000
IRAPA*	VEN	E ZULIA	126	138	2	136	0.014	0.986
MACOITA*	VEN	E ZULIA	126	78	0	78	0.000	1.000
PARIRI*	VEN	E ZULIA	126	74	0	74	0.000	1.000
SHAPARU*	VEN	E ZULIA	126	24	0	24	0.000	1.000

TABLE 9. THE TRANSFERRIN PHENOTYPES AND GENOTYPES. BECAUSE THE NON-C PHENOTYPES ARE SO MANY BUT YET INDIVIDUALLY SO RARE, GENE FREQUENCIES ARE GIVEN FOR C AND FOR ALL OTHERS COMBINED (EXPRESSED IN THE TABLE AS '1-C').

TRIBE OR GROUP	NATION	REGION	REF	SAMPLE SIZE	PHENOTYPES								GENE FREQUENCIES	
					B01	B2C	B01C	C	CDCHI	CD1	D1	DCHI	1-C	C
ACAWAIO*	GUY		022	84	0	0	0	84	0	0	0	0	0.000	1.000
AGUARUNA*	PRU	R MARANON	168	151	0	0	0	151	0	0	0	0	0.000	1.000
ALACALUF* 3	CHL	MAGELLAN	164	43	0	0	0	43	0	0	0	0	0.000	1.000
ATACAMENOS 1	CHL	ANTOFAGST	164	79	0	0	0	79	0	0	0	0	0.000	1.000
ATHABASCAN	USA	ALASKA	092	49	0	0	0	49	0	0	0	0	0.000	1.000
AWEIKOMA ANO*	BRZ	S CATARNA	237	37	0	0	0	37	0	0	0	0	0.000	1.000
AYMARA* 1	PRU	PUNO	093	56	0	0	0	56	0	0	0	0	0.000	1.000
AYMARA* 3	PRU	PUNO	168	71	0	0	0	71	0	0	0	0	0.000	1.000
AYMARA* 4	BOL	3 AREAS P	169	71	0	0	0	71	0	0	0	0	0.000	1.000
BARI*	VEN	W ZULIA	022	71	0	0	0	54	17	0	0	0	0.120	0.880
BRIBRI*	COS	SALITRE	237	38	0	0	0	33	5	0	0	0	0.066	0.934
CABECAR*	COS	UJARRAS	166	25	0	0	0	24	1	0	0	0	0.020	0.980
CAINGANG	BRZ	R G SUL	236	116	0	1	0	115	0	0	0	0	0.004	0.996
CAKCHIQUEL 1	GUA	L ALTITLN	165	150	0	0	0	142	0	8	0	0	0.027	0.973
CAKCHIQUEL* 2	GUA	SOLOLA	260	10	0	0	0	10	0	0	0	0	0.000	1.000
CAMPA*	PRU	R URUBAMB	168	93	0	0	0	93	0	0	0	0	0.000	1.000
CAYAPA*	ECD	R CAYAPAS	167	226	0	0	0	206	0	20	0	0	0.044	0.956
CHIAPANECA 1	MEX	CHIAPAS	165	40	0	0	0	38	0	2	0	0	0.025	0.975
CHIAPANECA 2	MEX	CHIAPAS	260	47	0	0	0	45	0	2	0	0	0.021	0.979
CHINANTECO	MEX	OAXACA	165	53	0	0	3	50	0	0	0	0	0.028	0.972
CHOCO*	PAN	DARIEN	166	74	0	0	0	74	0	0	0	0	0.000	1.000
CHOL 2	MEX	CHIAPAS	260	16	0	0	0	16	0	0	0	0	0.000	1.000
COLORADO* 2	ECD	S DOMINGO	167	36	0	0	0	36	0	0	0	0	0.000	1.000
CUNA* 2	PAN	S BLAS IS	166	174	0	0	0	174	0	0	0	0	0.000	1.000
GUAHIBO*	VEN	APURE	022	112	0	0	0	112	0	0	0	0	0.000	1.000
GUARANI*	BRZ	S CATARNA	237	30	0	0	0	30	0	0	0	0	0.000	1.000
GUAYMI*	PAN	N W COAST	166	204	0	0	0	180	0	24	0	0	0.059	0.941
IRAPA*	SEE	YUPA												
ISCONAHUA* 1	PRU	R UCAYALI	043	16	0	0	0	16	0	0	0	0	0.000	1.000
JIVARO* 2	ECD	ARAPICOS	167	221	0	0	0	220	0	1	0	0	0.002	0.998
KEKCHI 1	GUA	COBAN	165	162	0	0	0	153	0	9	0	0	0.028	0.972
KEKCHI* 2	BRH	TOLEDO	166	65	0	0	1	58	0	5	1	0	0.062	0.938
LACANDON* 1	MEX	CHIAPAS	260	31	0	0	0	31	0	0	0	0	0.000	1.000
LACANDON* 2	MEX	CHIAPAS	165	59	0	0	10	49	0	0	0	0	0.085	0.915
MACOITA*	SEE	YUPA												
MACUSHI*	GUY		022	116	0	0	0	116	0	0	0	0	0.000	1.000
MAKIRITARE* 1	VEN	S BOLIVAR	022	54	0	0	0	54	0	0	0	0	0.000	1.000
MAM 1	GUA	SAN JUAN	165	116	0	0	0	112	0	4	0	0	0.017	0.983
MAM 2	GUA	HUEHUETNG	260	27	0	0	0	27	0	0	0	0	0.000	1.000
MAYA 4	BRH	TOLEDO	166	212	0	0	0	195	0	16	1	0	0.042	0.958
NAVAJO 3	USA	ARIZ	203	230	0	1	16	213	0	0	0	0	0.037	0.963
PARIRI	SEE	YUPA												
PEMON* 3P	VEN	LA SABANA	022	96	0	0	0	96	0	0	0	0	0.000	1.000
PIAROA* 1	VEN	APURE	022	77	0	0	0	52	20	0	5	0	0.195	0.805
PIRO*	PRU	R URUBAMB	168	86	0	0	0	83	0	3	0	0	0.017	0.983
QUECHUA 2P	ECD	N AND CEN	167	192	0	0	0	165	0	27	0	0	0.070	0.930
QUICHE 1	GUA	S CENTRAL	260	94	0	0	1	92	0	1	0	0	0.011	0.989
RAMA	NIC	RAMA CAY	165	37	0	0	0	25	0	8	4	0	0.216	0.784
SANEMA* SHIRISHANA*	VEN	S BOLIVAR	022	74	0	0	0	74	0	0	0	0	0.000	1.000
SECOYA*	ECD	CUYABENDO	167	48	0	0	0	47	0	1	0	0	0.010	0.990
SHAPARU	SEE	YUPA												
SHIPIBO* 1	PRU	R UCAYALI	168	129	0	0	0	129	0	0	0	0	0.000	1.000
SHIPIBO* 2	PRU	R UCAYALI	043	70	0	0	0	70	0	0	0	0	0.000	1.000
SHIRISHANA*	SEE	SANEMA												

```
SUMO*                NIC  E COAST      165   108   0   0   0  108   0   0   0   0  0.000  1.000
TERRABAS*            COS  TERRABA      166    31   0   0   0   29   0   2   0   0  0.032  0.968
TICUNA*              PRU  R AMAZON     168   122   0   0   0  122   0   0   0   0  0.000  1.000
TOTONAC 2            MEX  VERA CRUZ    260    45   0   0   0   45   0   0   0   0  0.000  1.000
TZELTAL*             MEX  CHIAPAS      260    97   0   0   0   97   0   0   0   0  0.000  1.000
TZOTZIL* 1           MEX  CHIAPAS      165    79   0   0   0   75   0   4   0   0  0.025  0.975
TZOTZIL* 2           MEX  CHIAPAS      260    88   0   0   1   87   0   0   0   0  0.006  0.994
WAICA*               SEE   YANOMAMA
WAPISHANA            GUY               022   116   0   0   0  116   0   0   0   0  0.000  1.000
WARRAU*              VEN  ORINOCO D    022   123   0   0   0  121   2   0   0   0  0.008  0.992
XAVANTE*      3P     BRZ  MA GROSSO    247   521   0   0   0  521   0   0   0   0  0.000  1.000
YAGUA*               PRU  AMAZON       168     9   0   0   0    9   0   0   0   0  0.000  1.000
YANOMAMA*    10P     VEN  S BOLIVAR    014   430   0   0   0  428   0   0   2   0  0.005  0.995
  WAICA*             VEN  S BOLIVAR    022   136   0   0   0  136   0   0   0   0  0.000  1.000
YUPA*         2P     VEN  E ZULIA      020    91   0   0   0   38  38   0   0  15  0.374  0.626
  IRAPA* 2           VEN  E ZULIA      017    89   0   0   0   84   0   5   0   0  0.028  0.972
  MACOITA* 2         VEN  E ZULIA      017    42   0   0   0   38   0   4   0   0  0.048  0.952
  PARIRI*            VEN  E ZULIA      020    69   0   0   0   27  27   0   0  15  0.413  0.587
  SHAPARU*           VEN  E ZULIA      020    22   0   0   0   11  11   0   0   0  0.250  0.750
ZAPOTEC              MEX  OAXACA       260    80   0   0   2   78   0   0   0   0  0.013  0.987
ZOQUE                MEX  CHIAPAS      165    31   0   0   0   29   0   2   0   0  0.032  0.968
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TABLE 10. THE HAPTOGLOBIN PHENOTYPES AND GENF FRFQUENCIES. IN THE COMPUTATION OF THE LATTER, THE AHAPTOGLOBINEMIA PHENOTYPES AND THE 'OTHER' PHENOTYPES HAVE BEEN OMITTED.

TRIBE OR GROUP	NATION	REGION	REF	SAMPLE SIZE	PHENOTYPES						GENE FREQUENCIES		P
					1-1	2-1	2-1M	2-2	0	OTHER	HP1	HP2	
ACAWAIO*	GUY		018	87	38	42	0	5	2	0	0.694	0.306	
AGUARUNA*	PRU	R MARANON	168	151	30	78	0	43	0	0	0.457	0.543	
ALACALUF* 3	CHL	MAGELLAN	164	43	13	14	0	15	1	0	0.476	0.524	
APACHE* 2	USA	NEW MEX	261	98	34	47	0	17	0	0	0.587	0.413	
ATACAMENOS 1	CHL	ANTOFAGST	164	79	32	42	0	5	0	0	0.671	0.329	
ATHABASCAN 1	USA	ALASKA	032	202	35	98	0	63	3	3	0.429	0.571	
ATHABASCAN 2	USA	ALASKA	245	104	13	50	0	41	0	0	0.365	0.635	
ATHABASCAN 3	USA	ALASKA	032	284	51	89	0	136	8	0	0.346	0.654	
AWEIKOMA ANO*	BRZ	S CATRINA	237	112	53	40	1	17	1	0	0.664	0.336	
AYMARA* 1	PRU	PUNO	093	56	28	24	0	4	0	0	0.714	0.286	
AYMARA* 3	PRU	PUNO	168	71	36	26	0	8	1	0	0.700	0.300	
AYMARA* 4	BOL	3 AREAS P	169	71	36	26	0	8	1	0	0.700	0.300	
BRIBRI*	COS	SALITRE	237	38	1	13	0	24	0	0	0.197	0.803	
CARECAR*	COS	UJARRAS	166	25	4	14	0	7	0	0	0.440	0.560	
CAINGANG	BRZ	R G SUL	236	326	183	110	12	21	0	0	0.758	0.242	
CAKCHIQUEL 1	GUA	L ALTITLN	165	150	56	73	0	18	3	0	0.629	0.371	
CAKCHIQUEL* 2	GUA	SOLOLA	260	10	4	4	0	1	1	0	0.667	0.333	
CAMPA*	PRU	R URUBAMB	168	93	33	37	0	23	0	0	0.554	0.446	
CAYAPA* 2	ECD	R CAYAPAS	167	226	98	97	0	28	0	3	0.657	0.343	
CEARA	BRZ	NOT REPTD	105	24	4	13	2	5	0	0	0.477	0.523	
CHIAPANECA 1	MEX	CHIAPAS	260	47	21	20	1	5	0	0	0.674	0.326	
CHIAPANECA 2	MEX	CHIAPAS	165	35	8	16	0	11	0	0	0.457	0.543	
CHINANTEC 2	MEX	OAXACA	165	53	5	29	0	19	0	0	0.368	0.632	
CHOCO*	PAN	DARIEN	166	74	18	28	0	24	4	0	0.457	0.543	
CHOL 2	MEX	CHIAPAS	165	16	9	4	0	3	0	0	0.688	0.312	
COLORADO* 2	ECD	S DOMINGO	167	36	28	8	0	0	0	0	0.889	0.111	
CUNA* 2	PAN	S BLAS IS	166	174	19	83	4	60	8	0	0.373	0.627	
GUAHARIBO*	VEN	AMAZONAS	017	19	15	4	0	0	0	0	0.895	0.105	
GUAHIBO*	VEN	APURE	134	117	55	45	0	15	2	0	0.674	0.326	
GUARANI* 2	BRZ	S CATRINA	237	34	9	15	0	9	1	0	0.500	0.500	
GUAYMI*	PAN	N W COAST	166	204	80	91	0	33	0	0	0.615	0.385	
HAIDA	CAN	QUEEN C I	150	413	113	221	0	79	0	0	0.541	0.459	
IRAPA*	SEE	YUPA											
ISCONAHUA* 1	PRU	R UCAYALI	043	16	1	13	0	2	0	0	0.469	0.531	
JIVARO* 2	ECD	ARAPICOS	167	222	92	100	0	28	2	0	0.645	0.355	
KEKCHI 1	GUA	COBAN	165	162	74	66	0	22	0	0	0.661	0.339	
KEKCHI* 2	BRH	TOLEDO	166	65	24	26	0	13	2	0	0.587	0.413	
LACANDON* 1	MEX	CHIAPAS	260	31	24	4	0	0	3	0	0.929	0.071	
LACANDON* 2	MEX	CHIAPAS	165	59	49	9	0	0	1	0	0.922	0.078	
MACOITA*	SEE	YUPA											
MACUSHI*	GUY		018	119	20	54	0	27	18	0	0.465	0.535	
MAKIRITARE* 1	VEN	S BOLIVAR	019	85	12	30	0	37	6	0	0.342	0.658	
MAKIRITARE* 2	VEN	AMAZONAS	017	59	27	18	0	14	0	0	0.610	0.390	
MAM 1	GUA	SAN JUAN	165	116	31	55	2	24	4	0	0.532	0.468	
MAM 2	GUA	SAN JUAN	260	27	8	11	0	8	0	0	0.500	0.500	
MAYA 4	BRH	TOLEDO	166	212	92	94	0	26	0	0	0.656	0.344	
NAVAJO 3	USA	ARIZ	203	263	57	119	0	87	0	0	0.443	0.557	
PANARE*	VEN	W BOLIVAR	023	32	2	16	0	11	3	0	0.345	0.655	
PEMON* 3P	VEN	LA SABANA	019	214	40	113	0	49	12	0	0.478	0.522	
PIAROA* 1	VEN	APURE	019	98	58	33	0	6	1	0	0.768	0.232	
PIRO*	PRU	R URUBAMB	168	86	29	41	0	16	0	0	0.576	0.424	
QUECHUA 2P	ECD	N AND CEN	167	192	112	66	0	9	5	0	0.775	0.225	
QUICHE 1	GUA	S CENTRAL	260	94	36	46	0	12	0	0	0.628	0.372	
RAMA	NIC	RAMA CAY	165	37	19	12	0	6	0	0	0.676	0.324	
SANEMA*	SEE	YANOMAMA											

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SECOYA*             ECD   CUYABENDO      167     48   14   23    0   11    0    0    0.531   0.469
SHIPIBO* 1          PRU   R UCAYALI      168    129   54   58    0   17    0    0    0.643   0.357
SHIPIBO* 2          PRU   R UCAYALI      043     70   25   41    0    4    0    0    0.650   0.350
SUMO*               NIC   E COAST        165    108   31   49    1   27    0    0    0.519   0.481
TERRABAS*           COS   TERRABA        166     31    9   14    0    8    0    0    0.516   0.484
TICUNA*             PRU   R AMAZON       168    122   60   45    0   17    0    0    0.676   0.324
TLINGIT             USA   ALASKA         032     82   16   38    0   26    1    1    0.438   0.562
TOTONAC 2           MEX   VERA CRUZ      260     45   14   23    0    8    0    0    0.567   0.433
TZELTAL*            MEX   CHIAPAS        260     97   31   54    0   12    0    0    0.598   0.402
TZOTZIL* 1          MEX   CHIAPAS        165     80   35   38    0    7    0    0    0.675   0.325
TZOTZIL* 2          MEX   CHIAPAS        260     88   31   44    0   13    0    0    0.602   0.398
WAICA*              SEE    YANOMAMA
WAPISHANA           GUY                  018    120   34   58    0   22    6    0    0.553   0.447
XAVANTE*     3P     BRZ   MA GROSSO      247    521  114  254    1  139   13    0    0.475   0.525
YAGUA*              PRU   R AMAZON       168      9    2    3    0    3    1    0    0.438   0.562
YANOMAMA*   10P     VEN   S BOLIVAR      014    425  301  109    0   15    0    0    0.836   0.164
  SANEMA*           VEN   S BOLIVAR      017     19   15    4    0    0    0    0    0.895   0.105
  WAICA             VEN   S BOLIVAR      019    139   84   48    0    4    3    0    0.794   0.206
YARURO*             VEN   APURE          019    103   42   46    0   13    2    0    0.644   0.356
YUPA                VEN   E ZULIA        019     93   57   31    0    5    0    0    0.780   0.220
  IRAPA* 2          VEN   E ZULIA        017    129    1   51    0   77    0    0    0.205   0.795
  MACOITA* 2        VEN   E ZULIA        017     74   12   40    0   22    0    0    0.432   0.568
ZAPOTEC             MEX   OAXACA         260     80   22   40    1   17    0    0    0.532   0.468
```

TABLE 11. THE GM PHENOTYPES AND GENE FREQUENCIES, AS BASED ON TESTING WITH ANT1-GM(A), ANTI-GM(B), AND ANTI-GM(X) SERA.

TRIBE OR GROUP	NATION	REGION	REF	SAMPLE SIZE	PHENOTYPES A+X+B+	A+X-B+	A+X+B-	A+X-B-	GENE FREQUENCIES GMAX	GMA	GMAB	P
ACAWAIO*	GUY		083	84	0	0	65	19	0.524	0.476	0.000	
ATHABASCAN 1	USA	ALASKA	256	51	0	2	16	33	0.172	0.807	0.020	
AWEIKOMA ANO*	BRZ	S CATARNA	233	119	0	4	57	58	0.279	0.703	0.017	
CARIB 4	SUR		256	34	11	17	3	3	0.220	0.233	0.547	
EMERILLON	FRG		078	40	0	0	15	25	0.209	0.791	0.000	
GUARANI* 2	BRZ	S CATARNA	233	34	0	3	13	18	0.216	0.739	0.045	
MACUSHI*	GUY		083	116	0	0	53	63	0.263	0.737	0.000	
MAKIRITARE* 2	VEN	AMAZONAS	082	122	0	0	91	31	0.496	0.504	0.000	
MOCETONES*	BOL	AMAZONIA	223	76	0	1	55	20	0.476	0.517	0.007	
OAYANA*	FRG		078	97	0	1	36	60	0.207	0.788	0.005	
OYAMPI*	FRG		078	98	0	0	33	65	0.186	0.814	0.000	
PALIKOUR*	FRG		078	75	3	1	56	15	0.534	0.438	0.027	
WAICA*	SEE	YANOMAMA										
WAPISHANA	GUY		083	116	0	0	71	45	0.377	0.623	0.000	
XAVANTE*	BRZ	MA GROSSO	247	464	0	0	176	288	0.212	0.788	0.000	
YANOMAMA WAICA	VEN	S BOLIVAR	129	137	0	0	43	94	0.172	0.828	0.000	

TABLE 12. Data on genes that are probably absent in Indians who have no Caucasian or Negro admixture. Tabulation restricted to samples in which non-Indian ancestry is believed to be less than 5 per cent, as listed in Table 1. Samples with relatively high frequencies that may be due to technical problems are starred (*).

Lutheran

Among 32 samples tested with anti-Lu ^{a} alone, 28 contained no case of Lu(a+). They are reported in the following seventeen references: 33, 63, 126, 128, 129, 167, 168, 169, 170, 171, 173, 175, 176, 177, 178, 209, and 268. The 4 exceptions are:

Tribe or Group	*Area*	*Reference*	*Sample Size*	*Lu(a+)*
Aymara	Bolivia: Yungas	169	120	1
Cuna	Panama: San Blas Is.	178	388	2
Diegueño	U.S.A.: California	202	58	2
Kalapalo (2) (*)	Brazil: Mato Grosso	201	73	12
			639	

Among six samples tested with both anti-Lu ^{a} and anti-Lu ^{b}, no case of either Lu(a+) nor of Lu(b—) was revealed (References 33, 176, 177 and 178).

Kell

Among 40 samples tested with anti-K serum alone, 28 contained no positives, and may be considered to consist wholly of homozygous kk cases. They are reported in seventeen references: 42, 64, 74, 81, 90, 114, 132, 134, 145, 171, 175, 195, 223, 231, 262, 263, and 268. The 12 exceptions are:

Tribe or Group	*Area*	*Reference*	*Sample Size*	*K+*
Athabascan	U.S.A.: Alaska	3	206	1
Aweikoma	Brazil: Sta. Catarina	233	122	3
Carajá	Brazil: Sta. Isabel	108	35	7
Chol	Mexico: Chiapas	64	137	4
Cara	Mexico: Nayarit	64	96	6
Diegueño	U.S.A.: California	202	56	6
Huastec	Mexico: Vera Cruz	64	75	2
Huichol	Mexico: Jalisco	64	70	2
Kalapalo (2) (*)	Brazil: Mato Grosso	[illegible]	73	17
Maztec (1)	Mexico: Oaxaca	64	136	1
Nahua (3)	Mexico: Vera Cruz	64	141	3
Irapa (1)	Venezuela: E. Zulia	193	153	3
			1,300	

Among 74 samples tested with both anti-K and anti-k sera, 70 contained no case of Kell positive and may be considered to consist of homozygous kk individuals only. They are reported in references: 23, 28, 33, 43, 90, 123, 124, 126, 129, 130, 131, 134, 164, 167, 168, 169, 170, 171, 173, 176, 177, 178, 233 and 262. The 4 exceptions are:

Tribe or Group	*Area*	*Reference*	*Sample Size*	*K+*
Cherokee	U.S.A.: No. Carolina	209	78	1
Chippewa (*)	U.S.A.: Minnesota	159	161	24
Goajiro	Venezuela: W. Zulia	128	119	1
Navajo (4)	U.S.A.: Arizona	63	237	4
			595	30

All of the 30 K-positive cases were heterozygous Kk except two of the four Navajos, who were KK.

Wright

Among 41 samples tested with anti-Wr^a serum, not one contained a positively reacting case. References: 63, 91, 164, 167, 168, 169, 171, 173, 175, 176, 177, 178, 209, 231, and 233.

TABLE 12—*Continued*

Miltenberger

Among 40 samples tested with anti-Mi^{a} serum, only one contained positive cases. A sample of 232 Quechuas at Calderon, in northern Ecuador (ref. 167), contained four Mi^{a}+ individuals, two of whom were related as father and son. Relationship of the other two was not ruled out. The other 39 samples are reported in references 164, 167, 168, 169, 170, 171, 173, 175, 176, 177, 178 and 179.

Verweyst

Among 37 samples tested with anti-V^{w} serum, only one contained a positive case—in a sample of 162 Kekchi Mayans in Cobán, Guatemala (ref. 173), one individual was V^{w}+. The other references are 91, 164, 167, 168, 169, 171, 175, 176, 177, 178, and 209.

"V" (in Rh-hr system)

Among 28 samples tested with anti-V serum, only two samples contained positive cases. Among 12 Kekchis tested at Cobán, Guatemala (ref. 173), two were V+, and among 45 Totonacs (sample 2) at Vera Cruz, Mexico (ref. 170), one was V+. The other samples are referred to in 28, 167, 170, 171, 175, 176, 177, 178, and 209.

Berriens

Among 18 samples tested with anti-Be^{a} serum not one positive case was found. References are: 167, 171, 173, 176, 177, 178, and 209.

Sutter

Among 15 samples tested with anti-Js^{a} serum, only one contained a positive case—one individual in a sample of 119 Goajiros, sample no. 3, in western Zulia State in Venezuela (ref. 128). Other references are: 28, 126, 167, 171, 173, 176, 177, and 209.

TABLE 13. Tribes and groups estimated to have more than 5 per cent of non-Indian ancestry. Some tribes listed here will be found also among one or another of tables 1–11. Such duplication results from two or more samples having been drawn from the same tribe, whether by the same or by different workers, one or more samples indicating less than 5 per cent non-Indian ancestry and one or more indicating more than 5 per cent non-Indian admixture.

TRIBE OR GROUP	COUNTRY	REFERENCES
Alukuyana	Surinam	109
Andido	Argentina	205
Apache	U.S.A.	42, 90, 111
Arawak	Surinam	58
Araucanian (see also "Mapuche")	Chile	189, 196, 211, 237
Atacamenos	Chile	74, 164
Athabascan	U.S.A.	62, 87, 98, 250
Aymará	Bolivia; Peru	223; 225
Beaver	U.S.A.; Canada	62, 98, 156
Blackfeet	U.S.A.; Canada	5, 49, 110, 139 154, 155, 160, 162
Blood	U.S.A.; Canada	49, 139 154, 160, 162
Boruca	Costa Rica	177
Botocudo	Brazil	153
Cáchama	Venezuela	118, 119
Caddo	U.S.A.	99
Caingang	Brazil	76, 77
Canela	Brazil	22, 65, 146
Carajá	Brazil	106, 107
Caramanta	Colombia	11
Carib	Surinam	257
Carrier	Canada	98
Catawba	U.S.A.	210
Catio	Colombia	12
Catopaxi	Ecuador	242
Ceará	Brazil	17, 18
Chaguanca	Argentina	181, 254
Chamacoco	Argentina	181, 254
Chemehuevi	U.S.A.	42
Cherokee	U.S.A.	209, 250
Chiapaneca	Mexico	170, 260
Chibcha	Colombia	252
Chibcha	Peru	251
Chickasaw	U.S.A.	112, 250
Chinanteco	Mexico	165, 187
Chipaya	Bolivia	258
Chipewyan	U.S.A.	96, 98, 143
Chippewa	U.S.A.	52, 56, 159
Chiriguano	Argentina	181, 254
Choctaw	U.S.A.	57, 112, 250
Chol	Guatemala; Mexico	64, 170, 171, 260, 262
Chontol	Mexico	64
Chorotega	Nicaragua	165, 175
Chorotí	Argentina	182, 254
Colla	Argentina	182, 199, 254
Cora	Mexico	192
Cree	Canada and U.S.A.	51, 97, 143, 155, 156
Crow	U.S.A.	250
Cuna	Panama	72
Dogrib	U.S.A.	98
Emerillon	French Guiana	114
Flathead	U.S.A.	154, 155
Galibi	French Guiana	114
Goajiro	Venezuela and Columbia	128, 134, 152
Guambiano	Colombia	137
Guaraní	Brazil	216
Guatuso	Costa Rica	81
Guayquerí	Venezuela	134, 135
Haida	Canada	102, 150, 264
Hopi	U.S.A.	42, 250
Huichol	Mexico	192
Hupa	U.S.A.	104
Ica	Colombia	22, 134
Itonama	Bolivia	169
Itza Maya	Mexico	170, 260
Jicaque	Honduras	165, 174
Kansas	U.S.A.	112
Kariri	Brazil	207
Karok	U.S.A.	104
Katios	Colombia	12
Keresan	U.S.A.	4
Kitamaat	Canada	217
Klallam	Canada	102
Kuaiker	Colombia	136, 198
Kokonuko	Colombia	137
Kutchin	U.S.A.	145
Kwakiutl	Canada	89, 102, 217
Lacandon	Mexico	171
Lamistas	Peru	269
Lenca	Honduras	165, 174
Lloreos	Colombia	253
Makah	U.S.A.	102
Mapuche	Chile	74, 101, 184, 188, 189, 196, 211, 238, 239
Mataco	Argentina	7, 182, 254

TABLE 13—*Continued*

TRIBE OR GROUP	COUNTRY	REFERENCES
Maya	Br. Honduras and Mexico	95, 170, 171, 176, 218
Mextizo	Mexico	170
Micmac	Canada	33, 88
Miskito	Nicaragua	165, 175
Mixe	Mexico	64, 165
Mixtec	Mexico	64, 221
Mojave	U.S.A.	42
Montagnais	Canada	33, 219
Moré	Bolivia	169
Muckleshoot	Canada	102
Muskhogean	U.S.A.	112, 250
Nahua	Mexico	221
Naskapi	Canada	33, 183, 219
Navajo	U.S.A.	4, 6, 40, 42, 63, 191, 203
Nez Perce	U.S.A.	80
Nootka	Canada	102
Oajana	Surinam	187
Okanagon	U.S.A.; Canada	102, 103
Omaha	U.S.A.	157
Ona	Chile	211
Otomí	Mexico	187, 226, 229
Oyama	Surinam	257
Oyampi	French Guiana	114
Paéz	Colombia	10, 22, 131, 137
Panzaleo	Ecuador	241
Paraujano	Venezuela	16, 17, 127, 134
Pataxó	Brazil	208
Paya	Honduras	165, 174
Pawnee	U.S.A.	99
Pehuelche	Chile	189, 254
Pehuenche	Chile	74, 254
Penobscot	U.S.A.	3
Piegan	Canada	155
Pijáo	Colombia	213
Pilagá	Argentina	182, 199, 254
Pima	U.S.A.	42, 100
Ponca	U.S.A.	250
Pueblo	New Mexico	4, 5, 6, 39, 40
Puruhá	Ecuador	242
Quechua	Ecuador; Peru	3, 28, 93, 167, 168, 214, 223, 244
Quillacinga	Colombia	198
Quilleute	U.S.A.; Canada	102
Quinault	U.S.A.	104
Ramkokamekra	Brazil	65
Sac and Fox	U.S.A.	250
Salish	U.S.A.; Canada	62, 87, 89, 102, 141
Sapelo	Canada	219
Sarcee	Canada	50
Sekani	Canada	98
Senaca	U.S.A.	70
Seri and Yaqui	Mexico	192, 221
Shoshone	U.S.A.	250
Sibundoy	Colombia	198
Sioux	U.S.A.	154, 157, 250
Slave	Canada	87, 97, 98, 156
Stoney	Canada	50, 97, 98, 155
Subtiaba	Nicaragua	165, 175
Swinomish	U.S.A.; Canada	102, 103
Tacana	Bolivia	169
Tarahumara	Mexico	187, 221
Tarasco	Mexico	24, 221
Tariana	Brazil	31
Tewa (Pueblo)	U.S.A.	4
Tinneh	U.S.A.	206
Tlaxcalteca	Mexico	227
Tlingit	U.S.A.	62, 141, 187
Toba	Argentina	182, 199, 204, 254
Toltec	Mexico	270
Totonac	Mexico	170
Towa (Pueblo)	U.S.A.	5, 6
Trumai	Brazil	249
Tsimshian	Canada	102, 217
Tulalip	Canada	102
Tungurahua	Ecuador	242
Tuscarora	U.S.A.	185
Uru	Peru	267
Ute	U.S.A.	161, 163
Wichita	U.S.A.	99
Winnebago	U.S.A.	157
Wintun	U.S.A.	104
Yagan	Chile	75
Yakima	U.S.A.; Canada	102, 103
Yamana	Chile	74
Yaqui and Seri	Mexico	192, 221
Yurok	U.S.A.	104
Zapotec	Mexico	64, 170, 187, 260

TABLE 14. Samples of "mixed" Indians, i.e., Indian populations resulting from recent, known amalgamation of several groups, in which non-Indian ancestry appears not to exceed 5 per cent. Asterisk indicates apparent 100 per cent Indian ancestry. It is recognized that many tribes listed in Tables 1–11 may have resulted from unknown amalgamations in the past, but it is felt that for studies of genetic relationships and evolution of American Indian, every effort must be made to avoid the uncertainties introduced by recent fusion of long-separate groups.

COUNTRY	LOCALITY	TRIBE(S)	SAMPLE SIZE	REFERENCES
Argentina	Chaco *	Chunupi, etc.	55	182
	Chaco *	Chorote, Chulupie, Pilga, Toba	545	180
	Chaco *	Mataco, etc.	78	180
	Chaco	Toba, etc.	194	182
	Jujuy	Puna, etc.	209	205, 225
	Salta *	Chaguanco, Chamococo, Chiri	120	181
	Salta, Humahuaca *	Quechua	74	244
Brazil	Bahía, R. Colonia	Pataxos, etc.	23	208
	Mata Grosso, Southern *	Arawak, Carib, Tupi	587	66
	Mato Grosso, upper Xingu *	Arawak, Carib, Tupi, Trumai	89	248, 249
	R. G. do Sul, Missões *	Guarani	107	216
Colombia	Cauca Valley	Chibchan	236	73
	Tolima and Huila			
	Coyaima	Pijao	439	213
	Natagaima *	Pijao	281	123
Guatemala	Not stated	Seven regions	237	1
	Not stated *	Four communities	120	263
Ecuador	Near Quito *	Mixed	200	266
	Six provinces	Various	6,662	241, 242
Mexico	Yucatan	Mayan	223	95
	Chiapas, Tobasco and Yucatan	Mayan	124	218
	Various	Various	250	228
Paraguay	Chaco	Maca	111	265
Peru	Central highlands	Not stated	800	214
	Mocha and Laredo *	Not stated	200	8
	Puna	Aymara and Quechua	500	151
Surinam	Paramaribo	Arawak	88	60
	Paramaribo	Carib and others	177	60

REVIEWS: 39, 187, 230, 254